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AUTHOR Binder, Sue; Falk, Henry

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ABSTRACT

This document describes an agenda for the first 5 years of a comprehensive effort to eliminate childhood lead poisoning. In 1984, between 3 and 4 million children were estimated to have blood lead levels high enough to adversely affect intelligence and behavior. Lead in the home environment, especially lead-based paint, is the major source of lead poisoning. Benefits of preventing lead exposure include reduced medical and special education costs, increased productivity, and reduced infant mortality. The strategy for eliminating lead poisoning involves: (1) increasing the number of activities that lead to the prevention of childhood lead poisoning and the funding of such activities; (2) increasing the abatement of the use of lead-based paint in housing; (3) reducing children's exposure to lead in the environment; and (4) establishing national surveillance of children with elevated blood lead levels. Funds that would be needed to implement the strategy are described and recommendations for implementing the strategy are offered. Also discussed are research activities that would complement the strategy. Eight references are cited. Appendixes include: (1) an account of the effects of lead exposure on children and fetuses; (2) a description of the benefits and costs of preventing lead exposure; (3) a history of childhood lead poisoning prevention programs; (4) a list of organizations that promote awareness of lead poisoning; and (5) guidelines for the development of lead abatement programs. (BC)

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STRATEGIC PLAN FOR THE ELIMINATION OF CHILDHOOD LEAD POISONING

Developed for the Risk Management Subcommittee, Committee to Coordinate Environmental Health and Related Programs, U.S. Department of Health and Human Services.

February 1991







STRATEGIC PLAN FOR THE

ELIMINATION OF CHILDHOOD LEAD POISONING

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PREFACE

Three striking conclusions about childhood lead poisoning have emerged in the past several years: 1) the effects of exposure to even moderate amounts of lead are more pervasive and long-lasting than previously thought, 2) significant impairment of intelligence and neurobehavioral function is being reported at increasingly lower levels of lead in blood, and 3) millions of children in the United States have blood lead levels in this new range of concern. These findings have been reviewed in great detail elsewhere, and they are summarized here. They are not, however, the main subject of this report. The main subject is the public health response to our new understanding of childhood lead poisoning.

In this report, we set forth a strategy for eliminating childhood lead poisoning as a public health problem. Essential actions include increased support of programs that prevent childhood lead poisoning, increased abatement of lead-based paint and paint-contaminated dust in high-risk housing, reductions in other sources and pathways of lead exposure in children, and national surveillance for children with elevated blood lead levels. Finding and treating children with lead poisoning is critical, but not sufficient. Preventive actions must be taken to remove sources of lead in the child's environment before poisoning occurs.

Any plan to eliminate childhood lead poisoning in the United States must address the formidable problems posed by lead-based paint. Lead-based paint abatement has been neither widespread nor effective. Developing an effective, long-term lead-based paint abatement effort is probably the most critical factor in eliminating childhood lead poisoning. In this plan, approaches to developing this effort receive most attention. From a national viewpoint, the relative contribution from different sources of lead for children with high blood lead levels (that is, those with or likely to get lead poisoning) is different from that for children with low or moderate blood lead levels. For children with the highest blood lead levels, lead-based paint is a particularly important source. Strategies will need to be developed to focus abatement efforts on the highest priority groups (especially children with lead poisoning severe enough to require medical intervention, e.g., blood lead levels ≥ 25 ug/dL). Initial screening efforts will also have to be focused on areas where there are the greatest numbers of children with the highest blood lead levels (e.g., ≥ 25 ug/dL).

This plan also calls for reducing lead in other major sources and pathways of exposure. Ongoing regulatory and voluntary protective actions are important and must be strengthened. Lead is widely distributed in water, food, and air, but this lead is less likely to produce lead poisoning than lead in such concentrated sources as lead paint. Reducing the amount of lead in these environmental media, however, can have a profound effect on blood lead levels throughout the entire United States. This was demonstrated when lead was removed from gasoline. Reducing the amount of lead in



water, food, and air would help reduce the prevalence of lead poisoning and would help protect children with blood lead levels below the current definition of lead poisoning from adverse effects.

The role of exposure to soil lead, both directly and through the contribution of soil lead to lead in housedust, is still being investigated. The nature and degree of soil lead abatement that would be appropriate is unclear. The research needed to resolve the soil lead issues will take years. However, since so many children are being poisoned by lead-based paint, significant action on lead-based paint abatement should not be delayed while we await the results of research. Decisions on how to set up rational soil lead abatement programs will have to be made separately as more data become available. (However, it is critical not to further contaminate the soil during lead-based paint abatement efforts.)

We have made substantial progress in reducing exposure to lead; deaths and severe illness from lead poisoning (e.g., encephalopathy) are now rare. The results of recent studies indicate, however, that blood lead levels previously believed to be safe are adversely affecting the health of children. Millions of children in the United States are believed to have blood lead levels high enough to affect intelligence and development. The need to deal with preventing exposure at these lower levels will require increased efforts. The Administration is responding to this problem with increased resources. In FY 1992, the President's budget calls for \$14.95 million for the lead poisoning prevention program at the Centers for Disease Control and \$25 million for the new HOME abatement program of the Department of Housing and Urban Development.

In many ways, the tone of this report is one of understatement. The enormity of the task of eliminating childhood lead poisoning and the extensive public health benefits to be gained are very clear. This strategic plan is at best a first step. More detailed plans for implementation must follow, and then the work itself must be done.

Childhood lead poisoning has already affected millions of children, and it could affect millions more. Its impact on children is real, however silently it damages their brains and limits their abilities. Deciding to develop a strategic plan for the elimination of childhood lead poisoning is a bold step, and achieving the goal would 1 a great advance.



AUTHORS, CONTRIBUTORS, FEER REVIEWERS, AND ACKNOWLEDGEMENTS

PRINCIPAL AUTHORS

Sue Binder, M.D.

Centers for Disease Control
Center for Environmental Health and Injury Control
1600 Clifton Road, NE
Atlanta, Georgia 30333

Henry Falk, M.D., M.P.H.

Centers for Disease Control Center for Environmental Health and Injury Control 1600 Clifton Road, NE Atlanta, Georgia 30333

CONTRIBUTORS

FEDERAL

Max Lum, E.D.

Agency for Toxic Substances and Disease Registry Division of Health Education 1600 Clifton Road, NE Atlanta, Georgia 30333

Susanne Simon

Agency for Toxic Substances and Disease Registry Division of Health Education 1600 Clifton Road, NE Atlanta, Georgia 30333

James L. Pirkle, M.D., Ph.D.

Centers for Disease Control
Center for Environmental Health and Injury Control
1600 Clifton Road, NE
Atlanta, Georgia 30333

Joel Schwartz, Ph.D.

Environmental Protection Agency 401 M Street, SW, PM-221 Washington, D.C. 20460



CONTRIBUTORS (cont'd)

William McC. Hiscock
Health Care Financing Administration
Program Initiatives Branch
P.O. Box 26678
Baltimore, Maryland 21207

Jane Lin-Fu, M.D.

Health Resources and Services Administration Maternal and Child Health Bureau 5600 Fishers Lane Rockville, Maryland 20857

Donald T. Ryan

National Institute of Environmental Health Sciences 727 S. 26th Place Arlington, Virginia 22202

STATE AND LOCAL

Charles G. Copley
Office of the Health Commissioner
City of St. Louis
Department of Health and Hospitals
634 N. Grand
St. Louis, Missouri 63178

PRIVATE SECTOR

Anne Elixhauser, Ph.D.

Human Affairs Research Center, Battelle 370 L'Enfant Promenade, SW, Suite 900 Washington, D.C. 20024-2115

Mark S. Kamlet, Ph.D.

Camegie Mellon University
Department of Social and Decision Sciences
Pittsburgh, Pennsylvania 15213



V

CONTRIBUTORS (cont'd)

Paul A. Locke, Esq. Environmental Law Institute 1616 P Street, NW, Suite 200 Washington, DC 20036

Stephanie Pollack, Esq.
Conservation Law Foundation of New England
3 Joy Street
Boston, Massachusetts 02108-1497

PEER REVIEWERS

Anita S. Curran, M.D.

Robert Wood Johnson Medical School

University of Medicine and Dentistry of New Jersey

One Robert Wood Johnson Place

New Brunswick, New Jersey 08903

Richard J. Jackson, M.D.

California Department of Health Services

Hazard Identification and Risk Assessment Branch

2151 Berkeley Way, Room 619

Berkeley, California 94704-1011

James C. Keck
Baltimore City Health Department
Lead Poisoning Prevention Program
303 East Fayette Street
Baltimore, Maryland 21202

John F. Rosen, M.D.
Albert Einstein College of Medicine
Montefiore Medical Center
111 East 210th Street
Bronx, New York 10467



ACKNOWLEDGEMENTS

We appreciate the assistance of the following individuals who reviewed and commented on drafts of this report:

FEDERAL

Vernon N. Houk, M.D.

Centers for Disease Control Center for Environmental Health and Injury Control 1600 Clifton Road, NE Atlanta, Georgia 30333

Robert W. Amler, M.D.

Agency for Toxic Substances and Disease Registry 1600 Clifton Road, NE Atlanta, Georgia 30333

Elizabeth Cochran

Centers for Disease Control
Center for Environmental Health and Injury Control
1600 Clifton Road, NE
Atlanta, Georgia 30333

Gene Freund, M.D.

Centers for Disease Control National Institute for Occupational Safety and Healtl. 4676 Columbia Parkway Cincinnati, Ohio 45226

Teri Guilmette

Centers for Disease Control Center for Environmental Health and Injury Control 1600 Clifton Road, NE Atlanta, Georgia 30333

Daniel A. Hoffman, Ph.D.

Centers for Disease Control Center for Environmental Health and Injury Control 1600 Clifton Road, NE Atlanta, Georgia 30333



vii

Robert S. Murphy, M.S.P.H.

Centers for Disease Control National Center for Health Statistics Hyattsv''e, Maryland 20782

Daniel C. Fuschal, Ph.D.

Centers for Disease Control
Center for Environmental Health and Injury Control
1600 Clifton Road, NE
Atlanta, Georgia 30333

Jeffrey J. Sacks, M.D., M.P.H.

Centers for Disease Control Center for Environmental Health and Injury Control 1600 Clifton Road, NE Atlanta, Georgia 30333

Sandra C. Eberlee

Consumer Product Safety Commission 5401 Westbard Avenue Bethesda, Maryland 20816

Brian C. Lee, Ph.D.

Consumer Product Safety Commission 5401 Westbard Avenue Bethesda, Maryland 20816

Robert W. Elias, Ph.D.

U.S. Environmental Protection Agency Office of Research and Development Research Triangle Park, North Carolina 27711

Renate D. Kimbrough, M.D.

U.S. Environmental Protection Agency Office of the Administrator 401 M Street, SE, A-101 Washington, DC 20460

Ronnie Levin

U.S. Environmental Protection Agency Office of Research and Development 401 M Street, SE, H-8105 Washington, DC 20460



Dave E. Schutz, M.S., M.P.P.

U.S. Environmental Protection Agency Office of Toxic Substances 401 M Street, SE, TS-798 Washington, DC 20460

P. Michael Bolger, Ph.D., D.A.B.T.

U.S. Food and Drug Administration Division of Toxicological Review and Evaluation 200 C Street, SW, HFF-156 Washington, DC 20204

Ellis Goldman, M.C.P.

U.S. Department of Housing and Urban Development Office of Policy Development and Research 451 7th Street, SW Washington, DC 20410

Ronald J. Morony, P.E.

U.S. Department of Housing and Urban Development Office of Policy Development and Research 451 7th Street, SW Washington, DC 20410

Steve Weitz, M.U.P.

U.S. Department of Housing and Urban Development Office of Policy Development and Research 451 7th Street, SW Washington, DC 20410

Kathryn Mahaffey, Ph.D.

National Institute of Environmental Health Sciences 3223 Eden Avenue, Room 13 Cincinnati, Ohio 45267-0056

Mary McKnight

U.S. Department of Commerce National Institute of Standards and Tuchnology Gaithersburg, Maryland 20899



STATE AND LOCAL

Mary Jean Brown

Massachusetts Department of Public Health Childhood Lead Poisoning Prevention Program State Laboratory Institute 305 South Street Jamaica Plain, Massachusetts 02130

Mark Matulef, Ph.D.

Massachusetts Executive Office of Communities and Development Office of Program and Policy Development 100 Cambridge Street Boston, Massachusetts 02202

Lewis B. Prenney

Massachusetts Department of Public Health Childhood Lead Poisoning Prevention Program State Laboratory Institute 305 South Street Jamaica Plain, Massachusetts 02130

PRIVATE SECTOR

John B. Moran

Laborers' National Health and Safety Fund Occupational Safety and Health 905 16th Street, NW Washington, DC 20006

Herbert L. Needleman, M.D.

University of Pittsburgh School of Medicine 3811 O'Hara Street
Pittsburgh, Pennsylvania 15213

Margery Turner

The Urban Institute 2100 M Street, NW Washington, DC 20037



STRATEGIC PLAN FOR THE

ELIMINATION OF CHILDHOOD LEAD POISONING

EXECUTIVE SUMMARY

The U.S. Public Health Service Year 1990 and Year 2000 Objectives for the Nation aim for progressive declines in the numbers of lead-poisoned children in the United States, leading to the elimination of this disease. We believe that a concerted society-wide effort could virtually eliminate this disease as a public health problem in 20 years.

This plan, developed for the Committee to Coordinate Environmental Health and Related Programs of the U.S. Department of Health and Human Services, provides an agenda for the first 5 years of a comprehensive society-wide effort to eliminate childhood lead poisoning. The results and experience from this 5-year program will lead to the agenda for the following 15 years.

Lead is a poison that affects virtually every system of the body. Results of recent studies have shown that lead's adverse effects on the fetus and child occur at blood lead levels previously thought to be safe; in fact, if there is a threshold for the adverse effects of lead on the young, it may be close to zero.

Lead poisoning remains the most common and societally devastating environmental disease of young children. Enormous strides have been made in the past 5 to 10 years that have increased our understanding of the damaging, long-term effects of lead on children's intelligence and behavior. Today in the United States, millions of children from all geographic areas and socioeconomic strata have lead levels high enough to cause adverse health effects. Poor, minority children in the inner cities, who are already disadvantaged by inadequate nutrition and other factors, are particularly vulnerable to this disease.

Childhood lead exposure costs the United States billions of dollars from medical and special education costs for poisoned children, decreased future earnings, and mortality of newborns from intrauterine exposure to lead. Childhood lead poisoning continues in our society primarily because of lead exposure in the home environment, with lead-based paint being the principal high-dose source. It is the most important source for the highest-risk children (e.g., those with blood lead levels ≥ 25 ug/dL); preventive actions for such exposures should receive the highest priority.



Federal regulatory actions have significantly reduced or eliminated lead from many consumer products, including new paint and gasoline. Federal agencies continue to take actions further to reduce lead exposure from water, food, soil, air, and the workplace. Unfortunately, we are making little progress in eliminating the major source of high-dose lead poisoning, leaded paint from older housing.

In a new benefits analysis based on data from three studies, we estimate that the abatement of lead from all pre-1950 housing containing lead-based paint over the next 20 years would result in societal benefits of \$62 billion. This anticipated economic benefit is an additional incentive to society, since even if no economic benefits of abatement could be demonstrated, prevention of childhood lead poisoning would still be a worthwhile public health activity.

This plan contains recommendations for program and research activities. The four immediately essential elements of this effort are:

- 1) Increased childhood lead poisoning prevention programs and activities.
- 2) Effective abatement of leaded paint and lead paint-contaminated dust in high-risk housing.
- 3) Continued reduction of children's exposure to lead in the environment, particularly from water, food, air, soil, and the workplace.
- 4) Establishment of national surveillance for children with elevated blood lead levels.

Increased childhood lead poisoning prevention activities and national surveillance for elevated lead levels are essential parts of a national strategy to eliminate childhood lead poisoning for several reasons. Children should be screened for elevated blood lead levels so that affected children will receive appropriate medical attention and environmental follow-up. Initially, screening activities must focus on those areas with the greatest prevalence of children with the highest blood lead levels. Screening and surveillance data are also important for defining those areas in greatest need of intensive abatement programs and for evaluating the success of the national abatement program in eliminating this disease in targeted areas.

Effective lead-based paint abatement is essential for the elimination of childhood lead poisoning. Lead-based paint is the most concentrated source of lead to children and, historically, is the source most closely linked to lead poisoning in children. Many sources of lead, for example, food and soil, contribute to overall exposure of U.S. children to lead, but for children with the highest blood lead levels, that is, children with lead poisoning, lead-based paint is of particular importance.



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The development of a national strategy to abate lead-based paint is critical to the success of the effort to prevent lead poisoning. At present, far too few homes are being abated. To achieve maximum impact in the shortest time, lead-based paint abatement programs need to be closely linked with public health programs.

We recommend development of a national strategy for lead-based paint abatement that includes actions by both the private and the public sectors. Since the public health benefits and cost-effectiveness of lead-based paint and dust abatement are greatest in the housing most likely to contribute to lead poisoning, in the early years the emphasis should be on abating the housing units of affected children and the units likely to poison children in the near future.

To eliminate completely this disease, however, will require that all housing with lead-based paint eventually be addressed. A prioritized program will allow the highest-risk housing to be abated first, while enhanced programs, infrastructure, and technology continue to be developed. This national lead-based paint abatement program must include an evaluation component to ensure efficacy and safety for occupants as well as workers and their families.

This strategic plan focuses heavily on lead-based paint because of its key role in lead poisoning and because of the limited nature of previous efforts to reduce this source of lead. A national plan to eliminate childhood lead poisoning, however, must also focus on other widespread sources and pathways of lead exposure to children. Lead in water, food, soil, and air, in particular, may affect large numbers of children and may contribute to overall levels of lead in the population. Continued efforts to reduce these sources and pathways of lead exposure will result in lower average blood lead levels in the United States and will thereby further diminish the likelihood of lead poisoning developing even in children exposed to a high-dose source.

Childhood lead poisoning usually does not cause distinctive clinical symptoms, but the effects of childhood lead poisoning on intellectual and neurobehavioral functioning are pronounced and may persist for life. Furthermore, lead poisoning is entirely preventable. We understand the causes of lead poisoning and, most importantly, how to eliminate them. This plan establishes priorities and identifies steps toward that end.



SUMMARY OF CHAPTERS

Chapter 1. Introduction

Lead poisoning, the most common production is entirely preventable to entirely devastating environmental disease of young children, is entirely preventable to entirely devastating environmental disease of young children, is entirely preventable to entirely preventable t

Chapter 2. Health Effects of Lead and Lead Exposure

Lead is a dangerous and pervasa environmental poison, particularly harmful to fetuses and young children. The threshold for some of lead's health effects may be close to zero. The Agency for Toxic Substances and Disease Registry (ATSDR) estimated that between 3 and 4 million children in the U.S. (17% of all children) had blood lead levels above 15 ug/dL in 1984, levels high enough to adversely affect intelligence and behavior. Lead in the home environment, principally from lead-based paint, is the major source of lead poisoning. (See Appendix I for more details on the material in this chapter.)

Chapter 3. Benefits of Preventing Lead Exposure of Children and Fetuses

A benefits analysis was performed for this report, taking into account recent data on the effects of lead on children and fetuses. (In addition, an example of a cost-benefit analysis of a national lead-based paint abatement program, along with the detailed benefits analysis, appears in Appendix II.) For this analysis, the benefits of preventing children and fetuses from being exposed to lead are the costs that would have been associated with exposure had it occurred. On the basis of this analysis, the average benefits of preventing a child's blood lead level from exceeding 24 ug/dL (the level at which medical evaluation is necessary) are \$4,631 for avoided medical and special education costs. For all children, including those with blood lead levels below 25 ug/dL, the average increased wages to be expected from preventing each 1 ug/dL increase in a child's blood lead level are \$1,147. The average benefits of preventing a 1 ug/dL increase in the blood lead level of a pregnant woman are \$300. Based on data from three programs (see Appendix II), the benefits of abating all pre-1950 housing with lead-based paint over a 20-year period would be \$62 billion, discounted to the present.

Chapter 4. Program Agenda

The four essential program components of a strategy to eliminate childhood lead poisoning are:

1) Increased childhood lead poisoning prevention programs and activities.



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- 2) Increased abatement of leaded paint and paint-contaminated dust in housing.
- 3) Continued reduction of children's exposure to lead in the environment, particularly from water, food, air, soil, and the workplace.
- 4) Establishment of national surveillance for children with elevated blood lead levels.

Increased childhood lead poisoning prevention activities include both funding of public lead poisoning prevention programs and increased awareness and action by private physicians. Increased abatement should also result from a combination of efforts by the private and public sectors. Before we can safely and effectively conduct as many abatements as are needed, the infrastructure for abatement must be developed. (Appendix V discusses infrastructure development in more detail.) Other environmental sources of lead should also continue to be addressed as part of the strategic plan; reductions of lead in water, food, soil, air, and the workplace are of most importance National surveillance for elevated blood lead levels is needed to target areas requiring increased lead poisoning prevention activities and abatement, to track our progress in eliminating childhood lead poisoning, and to evaluate lead exposure in abatement workers and workers in other lead-contaminated environments.

Chapter 5. Research Agenda

Research activities to complement the four essential program components are described in this chapter.

Chapter 6. Funds Needed for Implementation of the Strategic Plan

Significant Federal, State, local, and private resources must be committed to meet the 5-year goals. Preliminary estimates indicate that as much as \$974 million in combined resources may be required to implement the first 5 years of this Strategic Plan.

Chapter 7. Summary of Recommendations

The five most urgent recommendations of this plan include increased prevention activities, increased abatement, reduced exposure to other sources of environmental lead, national surveillance, and research.



CHAPTER 1. INTRODUCTION

INTRODUCTION

- CHILDHOOD LEAD POISONING IS EXTREMELY WIDESPREAD.
- ALTHOUGH SUBSTANTIAL PROGRESS HAS BEEN MADE IN THE PAST 20 YEARS, KEY ENVIRONMENTAL SOURCES OF LEAD REMAIN.
- THIS DOCUMENT PRESENTS A STRATEGIC PLAN FOR THE E'IMINATION OF CHILDHOOD LEAD POISONING.

Lead poisoning remains the most common and societally devastating environmental disease of young children. Millions of U.S. children from all geographic areas and socioeconomic strata have blood lead levels high enough to be associated with adverse health effects. Poor, minority children in the inner cities, who are often already disadvantaged by inadequate nutrition and other factors, are particularly vulnerable to this disease. The pervasiveness of childhood lead poisoning was well described in The Nature and Extent of Childhood Lead Poisoning in Children in the United States: a Report to Congress, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR, 1988).



Childhood lead poisoning is entirely preventable. We understand the causes of lead poisoning and, most importantly, how to eliminate them. We believe that a concerted societal effort could virtually eliminate this disease as a public health problem in 20 years.

Important progress has been made in reducing some sources of lead in the past 20 years. Federal regulatory actions have significantly reduced or eliminated lead from many consumer products, including new paint and gasoline. Voluntary programs, such as the work by the Food and Drug Administration (FDA) with can manufacturers to reduce lead in canned food, have also been highly successful in reducing exposure to lead. Federal agencies continue to take actions to further reduce lead exposure from water, food, air, and the workplace. Unfortunately, limited progress has been made in eliminating lead-based paint from older housing—the major source of high-dose lead poisoning in children. Abatement of lead-painted homes is an essential part of both the prevention of childhood lead poisoning and the treatment of poisoned children.

LEAD-BASED PAINT ABATEMENT IS AN INTEGRAL PART OF THE TREATMENT OF CHILDHOOD LEAD POISONING AND THE PREVENTION OF NEW CASES. WE HAVE MADE LITTLE PROGRESS IN ELIMINATING LEAD-BASED PAINT IN OLDER HOMES AS A CAUSE OF CHILDHOOD LEAD POISONING.

The lack of progress in eliminating childhood lead poisoning is due to several factors. For example, lead poisoning has been improperly considered by many to be a disease of the poor that could be remedied by better housekeeping and childrearing; another source of confusion is that many people believe the disease was eliminated when the manufacture of lead-based paint for residential use was banned. The logistical difficulties and high costs of abating lead-based paint in homes have also been a major problem.



During the past 20 years, severe, symptomatic lead poisoning in children (e.g., encephalopathy with coma) has been markedly reduced. However, new and increased knowledge and awareness of the health effects of exposure to lead in childhood, especially at lower levels once considered safe, have dramatically increased concern about this problem in recent years. The fact that childhood lead poisoning is a societally devastating, yet totally preventable disease has focused attention on the need for a strategic plan to eliminate it.

DEATHS AND ACUTE, SEVERF ILLNESS FROM LEAD POISONING ARE NOW RARE. HOWEVER, WE NOW KNOW THAT LARGE NUMBERS OF CHILDREN MAY SUFFER ADVERSE HEALTH EFFECTS AT BLOOD LEAD LEVELS THAT WERE ONCE CONSIDERED SAFE.

Several recent government documents have extensively reviewed health and environmental data related to childhood lead exposure (ATSDR, 1988; Environmental Protection Agency, 1986). This strategic plan discusses these data briefly, but focuses on a detailed benefits analysis and major agenda items. The plan consists of chapters on exposure to lead and its effects on children and fetuses, a benefits analysis of reducing lead exposure, a program agenda, a research agenda, and a discussion of the funds needed for implementation. Several appendices provide the background and justification for the material in the plan.

This document has been developed at the request of Dr. James O. Mason, Assistant Secretary for Health, U.S. Department of Health and Human Services, for the Committee to Coordinate Environmental Health and Related Programs. It has been developed with the help of contributors from other Federal, State, and local agencies and the private sector. It does not, however, necessarily reflect the policies of these individuals and agencies.



CHAPTER 2. HEALTH EFFECTS OF LEAD AND LEAD EXPOSURE (See Appendix I for more details on material in this section.)

EFFECTS OF EXPOSURE

HEALTH EFFECTS

LEAD AFFECTS EVERY SYSTEM IN THE BODY.

EFFECTS ON INTELLIGENCE AND BEHAVIOR ARE MOST IMPORTANT.

LEAD EXPOSURE

CHILDREN ARE EXPOSED TO LEAD FROM MANY SOURCES AND PATHWAYS.

LEAD-BASED PAINT IS THE SOURCE OF GREATEST CONCERN.

Lead is an extremely dangerous and pervasive environmental poison. In 1984, at least 3 to 4 million children in the United States (17% of all children) had blood lead levels high enough to cause neurobehavioral and other adverse health effects (ATSDR, 1988). The large number of children with blood lead levels in the toxic range shows that existing environmental lead levels in the United States provide no margin of safety for the protection of children.

The risks of lead exposure are not based on theoretical calculations. They are well known from studies of children themselves and are not extrapolated from data on laboratory animals or high-dose occupational exposures. Whereas conservative approaches are used to estimate risk from low level exposures to many chemicals, especially carcing gens, this is not the case for lead.



ADVERSE EFFECTS OF LEAD ON CHILDREN AND THE FETUS

Neurobehavioral

Decreased intelligence
Developmental delays
Behavioral disturbances
Seizures (at very high levels)
Coma (at very high levels)

- Growth
 Decreased stature
- Endocrinologic
 Altered vitamin D metabolism
- Hematologic
 Elevated erythrocyte protoporphyrin levels
 Anemia
- On the fetus

Decreased gestational weight Decreased gestational age Miscarriage and stillbirth (at very high levels)

Lead is a poison that affects virtually every system in the body. It is particularly harmful to the developing brain and nervous system; therefore, lead exposure is especially devastating to fetuses and young children.

Very severe lead exposure (blood lead levels ≥ 80 ug/dL) can cause coma, convulsions, and even death. It is currently estimated that there are about 250,000 children under 6 years of age whose blood lead is 25 ug/dl and greater. The adverse effects on these children are great. They need to be identified as soon as possible to remove them from the source of lead and provide appropriate medical care. This is the highest priority. Blood lead levels as low as 10 ug/dL, which usually do not cause distinctive symptoms, are associated with decreased intelligence and slower neurobehavioral development. Other effects that begin at blood lead levels as low as 10 ug/dL include behavioral disturbances, reduced stature, and effects on vitamin D metabolism. Maternal and cord blood lead levels of 10 to 15 ug/dL appear to be associated with reduced gestational age

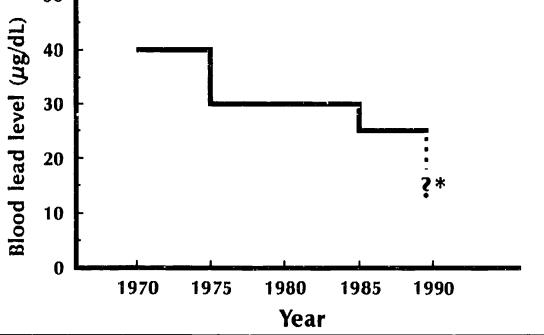


and reduced weight at birth (ATSDR, 1988). Blood lead levels of 10 ug/dL and above at age 2 years have been shown to result in a reduction of the General Cognitive Index at age 57 months. Most of the children studied had blood levels below 15 ug/dL (Bellinger, 1991). Although researchers have not yet completely defined the impact of blood lead levels <10 ug/dL on central nervous system function, it may be that even these levels are associated with adverse effects that will be more clear as our research instruments become better.

The neurobehavioral effects of childhood lead exposure appear to be longlasting.

In a recent long-term follow-up study (Needleman, 1990), for children who had been exposed to mederate lead levels in preschool years, the odds of those children dropping out of high school were seven times higher, and the odds of a significant reading disability were six times higher than for children exposed to lower lead levels. In addition, the children exposed to higher lead levels had lower class standing, increased absenteeism, and lower vocabulary and grammatical-reasoning scores, even after investigators controlled for other covariates. The apparent persistence or irreversibility of many of lead's neurobehavioral effects intensifies concern over exposure of fetuses and children to lead.

Blood lead levels considered elevated by CDC 50



*Currently undergoing revision

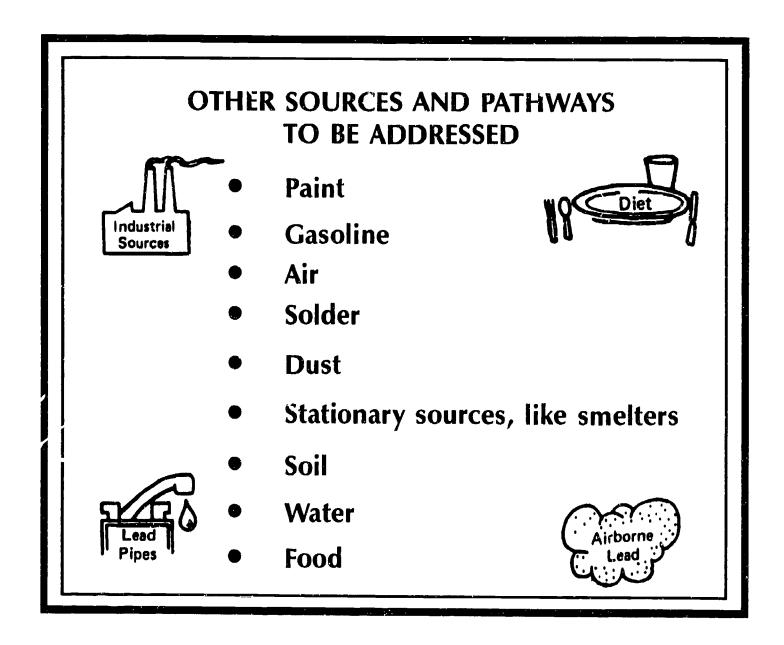


Studies on the health effects of lead over the past 20 years have produced a consistent trend: the more that is learned about lead's effects on children and fetuses, the lower the blood lead level at which adverse effects can be documented. In the first half of the 20th Century, medical care providers were concerned about blood lead levels ≥ 80 ug/dL; by the 1960s, they were concerned about levels ≥ 60 ug/dL; in the 1970s, as studies began showing effects at lower and lower levels, the level of concern was at 40 ug/dL; and by the middle 1980s, it was lowered to 25 ug/dL. A current reassessment will likely place the level at which interventions are recommended at 10-15 ug/dL. Blood lead levels formerly considered safe have now been clearly associated with adverse effects. If there is a threshold for lead's effects on health, it is probably near zero.

The definition of childhood lead poisoning requires a blood lead level of $\geq 25 \ \mu g/dL$. This definition is being reconsidered and the blood lead level is being revised downward.

The current definition of childhood lead poisoning requires a blood lead level ≥ 25 ug/dL (CDC, 1985). This definition is being reevaluated and, as a result of recent research on the effects of low-level lead exposure in children, it will undoubtedly be lowered to 10-15 ug/dL. A Federal advisory committee is currently meeting and working on these changes.





Lead has some unusual characteristics that cause special concern about exposure. First, lead deposited in the environment does not biodegrade; it remains there and accumulates. Second, lead exposure is pervasive, sparing no segment of the U.S. populace. Third, lead accumulates over months and years in the bodies of children. Therefore, chronic exposure to small sources of lead can result in a large long-term accumulation in a child, increasing that child's risk of adverse health effects. During pregnancy, a woman's bone lead stores may be mobilized, exposing the fetus to lead. Thus, childhood lead exposures in one generation may result in prenatal exposure in the next generation.



Children are exposed to lead from many sources (for example, paint, gasoline, solder, and stationary sources like smelters) via multiple pathways (for example, air, dust, soil, water, and food). A child's particular environment determines the relative importance of each source and pathway.

Today, lead-based paint is the source of greatest public health concern. It is the most common cause of high-dose lead exposure. Exposure occurs not only when children ingest chips and flakes of paint (which often contain as much as 50 percent lead by weight), but also, and probably more commonly, when children ingest lead-based paint-contaminated dust and soil during normal mouthing activities.

In the mid-1980s, about 13.6 million children under 7 years of age lived in homes with lead-based paint. An estimated 1.8 to 2.0 million children lived in deteriorated lead-painted housing with unsound paint (for example, peeling paint and other damage to walls), which placed them at high risk of excessive lead exposure from this source; about 1.2 million of these children were estimated to have blood lead levels above 15 ug/dL, mainly because of exposure to lead paint (ATSDR, 1988). ATSDR has assessed existing lead paint in U.S. housing and public buildings to be an "untouched and enormously serious problem."

LEAD-BASED PAINT IS THE SOURCE OF GREATEST PUBLIC HEALTH CONCERN. OTHER SOURCES OF LEAD ALSO CAN BE IMPORTANT CONTRIBUTORS TO CHILDREN'S BLOOD LEAD LEVELS.

Estimates of numbers of children exposed to other sources and pathways of lead appear in Appendix I. The removal of lead from gasoline during the last decade, as well as reductions in other widespread sources and pathways such as lead in food, has contributed to a major drop in the mean blood lead levels of children. By lowering the average, or baseline, level of lead in children, the risk of lead poisoning is reduced, even from exposure to concentrated sources such as lead paint, because higher doses are necessary to produce lead poisoning. It is, therefore, important to continue to reduce children's exposure to lead from air, water, food, soil, and the workplace; there will also be occasions where these sources and pathways result in lead poisoning. Efforts to reduce these exposures are not a substitute for lead-based paint abatement, however, because in the geographic areas where lead-based paint and dust are a prominent hazard, they alone can, as noted above, produce childhood lead poisoning.



CHAPTER 3. BENEFITS OF PREVENTING LEAD EXPOSURE OF CHILDREN AND FETUSES (The methods and assumptions on which this benefits analysis are based are detailed in Appendix II. Numerical estimates are included only for those benefits which we believe are defensible by good, quantative data. Not factored in the benefits analysis are those which are not able to be quantified. Appendix II also contains a detailed cost-benefit analysis, in which the benefits of reducing lead exposure are compared to the costs of lead-based paint abatement, based on the three currently available studies for which we had data both on the costs of abatement and the resultant changes in blood lead levels.)

BENEFITS OF PREVENTING LEAD EXPOSURE

THE BENEFITS WE QUANTIFIED ARE:

- REDUCED MEDICAL COSTS
- REDUCED SPECIAL EDUCATION COSTS
- INCREASED FUTURE PRODUCTIVITY
- REDUCED INFANT MORTALITY

Lead exposure in U.S. children is estimated to cost society billions of dollars a year (for example, Levin, 1986). These estimates have included costs of medical care, special education and institutionalization, and decreases in productivity and lifetime earnings resulting from impaired cognition.



For this strategic plan, we have developed a new benefits analysis. The analysis is detailed in Appendix II and is focused on the benefits of preventing exposure to lead in children and fetuses. The benefits of reducing lead exposure in persons already being exposed are likely to be substantial, but they are difficult to quantitate. For example, we do not know how long lead levels must be elevated before a child develops cognitive deficits or before these deficits become irreversible. We, therefore, did not include already exposed individuals in the main benefits analysis.

For this analysis, the benefits of preventing children and fetuses from being exposed to lead are the avoided costs that would have been associated with exposure. The four benefits for which we provide monetary values for prevention are 1) reduction in medical care costs of poisoned children, 2) reduction in special education costs for poisoned children, 3) reduction in future lost productivity from cognitive deficits in children, and 4) reduction in neonatal mortality from prenatal lead exposure. These are but a few of the benefits of preventing lead exposure. We did not evaluate the benefits related to children's stature, hearing, vitamin D metabolism, and blood production; the benefits of preventing the effects of lead on adults; or nonhealth-related benefits such as reduced personal injury court cases and improved property values.

The benefits we evaluated fall into two categories: 1) The first category consists of benefits achieved only for children whose blood lead levels are prevented from rising above a certain threshold; avoided medical and special education costs are estimated only for those children prevented from developing blood lead levels ≥25 ug/dL. 2) The second category consists of the benefits of preventing increased blood lead levels in children no matter what their initial levels are. For example, intellectual deficits result over a broad range of blood lead levels. Estimates of costs saved by reducing the effects of lead on intellectual functioning were made for preventing increases of 1 ug/dL in blood lead level, regardless of the starting blood lead levels. The benefits of reducing maternal blood lead levels, which results in decreased infant mortality, are included in the second category.

Average benefits of preventing

Blood lead levels from rising above 24 μ g/dL:

Avoided medical costs \$1,300 per child Avoided special education costs \$3,331 per child

A 1 μ g/dL increase in blood lead level, regardless of starting blood lead level:

Increased lifetime earnings Reduced infant mortality \$1,147 per μ g/dL per child \$ 300 per μ g/dL per newborn



The average total medical cost avoided by preventing a child's blood lead level from rising above 24 ug/dL is \$1,300 per child. (This amount is lower than the cost per episode for chelation, because not all children with elevated blood lead levels will be chelated.) On the average, \$3,331 per child is saved in special education costs. By preventing an increase of 1 ug/dL in a child's blood lead level, a net present value benefit of \$1,147 per child from increased future income is saved. Clearly, the greater the prevented increase in blood lead level, the greater the benefits; for the individual child, preventing the blood lead level from exceeding 24 ug/dL results in maximum benefits. Preventing a 1 ug/dL increase in the blood lead level of a pregnant woman saves an average of \$300 from reduced infant mortality. (Assumptions used in quantifying these benefits, including the monetary benefits of preventing infant mortality, are in Appendix II.)

EXAMPLE:

The benefits of preventing a child's blood lead level from rising from 24 μ g/dL to 34 μ g/dL are:

11,470
11,470 16,101

When these figures for the individual (average) child are applied nationally, the benefits of eliminating childhood lead poisoning are striking. For example, based on data from 3 programs (See Appendix II), the benefits of abating all pre-1950 housing with lead-based paint over a 20-year period would be \$62 billion, discounted to the present.

SEE APPENDIX II FOR

- Detailed benefits analysis
- An illustration of the cost-benefit analysis for abatement of lead-based paint in all pre-1950 housing
- Sensitivity analyses



THE PROGRAM AGENDA FOR THE NEXT 5 YEARS CONTAINS FOUR MAIN ITEMS

- INCREASED CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES
- INCREASED ABATEMENT OF LEADED PAINT AND PAINT-CONTAMINATED DUST IN HOUSING
- REDUCTIONS IN OTHER SOURCES AND PATHWAYS OF LEAD EXPOSURE
- NATIONAL SURVEILLANCE

The program agenda for the first 5 years of the effort to eliminate childhood lead poisoning has four essential components: 1) increased childhood lead poisoning prevention activities, 2) increased abatement of leaded paint and paint-contaminated dust in housing, 3) continued efforts to reduce other widespread sources and pathways of lead exposure, and 4) national surveillance for elevated blood lead levels. Education and public awareness are essential to success in implementing all of these components.

PROGRAM AGENDA ITEM 1. INCREASED CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES

INCREASED CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES MEANS

- INCREASED FUNDING FOR FEDERALLY-SUPPORTED PROGRAMS
- OTHER EFFORTS TO INCREASE SCREENING AND EDUCATION
- DEVELOPMENT OF INFRASTRUCTURE TO SUPPORT INCREASED PROGRAMS

For this document, childhood lead poisoning prevention activities are defined as the screening of children for elevated blood lead levels, referral of poisoned children for medical and environmental interventions, and education about childhood lead poisoning. Such education is not limited to increasing public and medical provider awareness of lead poisoning. It also includes the education of children with elevated blood lead levels and their families about nutritional and other interventions. Expansion of childhood lead poisoning prevention activities should first focus on those children with the highest blood lead levels (e.g., blood lead levels $\geq 25 \text{ ug/dL}$).



Most children with lead poisoning are never identified.

An estimated 250,000 children had blood lead levels >25 ug/dL in 1984 (ATSDR, 1988). (More up-to-date estimates will be available in the next couple of years from the Third National Health and Nutrition Examination Survey.) Available data indicate that the majority of such lead-poisoned children are never identified. The screening of children for elevated blood lead levels must be increased so that poisoned children can receive appropriate medical attention and environmental follow-up. (Environmental follow-up varies widely among programs and includes the measurement of lead in paint and often other potential media and interventions to prevent further exposure.) Screening data are also important for defining those areas in greatest need of intensive abatement programs and for evaluating the success of abatement programs in eliminating this disease in targeted areas.

Federally-Supported Childhood Lead Poisoning Prevention Programs

FEDERALLY-SUPPORTED PROGRAMS

- Federal programs began in 1972.
- Programs are administered by several agencies.
- Programs are directed at children at highest risk for lead poisoning.
- Programs screen only a small percentage of children at risk.



The history of childhood lead poisoning prevention programs in the United States is summarized in Appendix III. State and local childhood lead poisoning prevention programs perform many functions. They screen large numbers of children for lead poisoning and accept referrals of poisoned children from other practitioners for follow-up. They ensure that appropriate investigations are conducted of the homes and other environments of poisoned children. They may issue orders for abatement and may work with other government agencies to have abatements done. They also make sure that children receive appropriate medical treatment and that any other young children in the family or household are screened for lead poisoning. They educate parents and health care providers about lead poisoning and ways of preventing it.

Door-to-door screening in high-risk neighborhoods generally is the most productive method of identifying children with lead poisoning. Early in the 1970s, community outreach and door-to-door screening efforts were an essential component of programs. However, these activities are labor-intensive and costly. Consequently, most programs now screen children in fixed-site facilities.

The national effort to identify children with lead poisoning and abate the sources of lead in their environments began with the passage of the Lead-Based Paint Poisoning Prevention Act of 1971. Federally-funded screening began in Fiscal Year 1972 with blood lead testing, but in 1975 the Centers for Disease Control (CDC) recommended screening with erythrocyte protoporphyrin (EP) instead. (EP levels are elevated in the presence of elevated blood lead levels. Although useful for identifying children with blood lead levels above about 30 ug/dL and for detecting iron deficiency, EP is not a sensitive test for identifying children with blood lead levels below 25 ug/dL.) For most of the early years of this program, Federal funds appropriated under this Act were administered by the CDC. More than \$89 million were distributed, and over a quarter of a million children were identified with lead poisoning and received referrals for environmental and medical intervention. The improvement in the health status of children identified with lead poisoning in this program was documented in an evaluation by F.D. Kennedy (1978).

Current major sources of Federal funding for screening programs are the Maternal and Child Health (MCH) Block Grant Program, administered by the Health Resources and Services Administration (HRSA), and the Categorical Grant Program, administered by the CDC.



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SOURCES OF FEDERAL FUNDING

- Maternal and Child Health Block Grants
- Centers for Disease Control Categorical Grant Program
- Early and Periodic Screening, Diagnostic, and Treatment Program (EPSDT)
- Supplemental Food Program for Women, Infants, and Children (WIC)
- Head Start

The MCH Block Grants serve as the principal means of Federal support to States to maintain and improve the health of mothers and children, including children with special needs. These grants are made to State health agencies to assure access to MCH services, especially for those with low income who live in areas with limited health services, and to reduce the incidence of preventable diseases and handicapping conditions in children. After assuming administrative responsibility for the Lead-Based Paint Poisoning Prevention Act in Fiscal Year 1982, HRSA issued a policy statement to all State MCH and Crippled Children's Services recommending routine periodic EP screening for all preschool children. Although not all States use MCH block grant funds for childhood lead screening, a 1984 survey indicated that 40 States, the District of Columbia, and Puerto Rico had screening activities.



The CDC Categorical Grant Program was authorized by the Lead Contamination Control Act of 1988. This program provides for childhood lead screening by State and local agencies, referral of children with elevated blood lead levels for treatment and environmental interventions, and education about childhood lead poisoning prevention. Money for this program was first appropriated in FY 1990. The President's budget for FY 1992 contains \$14.95 million for this program, an increase of \$7.16 million from FY 1991.

Other government-funded child health programs also conduct some childhood lead screening. These programs include Medicaid's Early and Periodic Screening, Diagnostic, and Treatment Program (EPSDT); the Supplemental Food Program for Women, Infants, and Children (WIC); and Head Start.

EPSDT is a comprehensive prevention and treatment program available to Medicaid-eligible persons under 21 years of age. In 1989, of the 10 million eligible persons, more than 4 million received initial or periodic screening health examinations. These are provided at a variety of sites (for example, physician offices, public health clinics, and community health centers) by private or public sector providers. Screening services, defined by statute, must include a blood lead assessment "where age and risk factors indicate it is medically appropriate." (The requirements for a blood lead assessment are not further defined.) In addition, the EP test is recommended for children ages 1 to 5 years to screen for iron deficiency. Because this test is also useful in identifying children with blood lead levels ≥ 25 ug/dL, many children being screened for iron deficiency are screened for lead poisoning at the same time. The guidelines for States indicate that environmental investigations for lead-poisoned children are covered under EPSDT, although abatement is not. However, specific criteria for screening and the determination of what Medicaid will cover are decided on a State-by-State basis. Thus, many States do not conduct much screening or do not pay for environmental investigations for poisoned children. National data are not available on the numbers of children screened for lead poisoning through EPSDT, since State-reported Medicaid performance and fiscal data are not broken down to such specific elements.

The U.S. Department of Agriculture's WIC program serves pregnant and postpartum women and children under 5 years of age in low-income households. Program benefits include supplemental food, nutrition education, and encouragement and coordination for the use of other existing health services. As of March 1988, an estimated 1.63 million children ages 1 to 4 years were participating in WIC. Although children must undergo a medical or nutritional assessment or both to be certified to receive benefits, Federal WIC regulations permit States to establish their own requirements for WIC certification examinations. These regulations permit the use of an EP test for certification and define lead poisoning as a nutritionally-related medical condition that can be the basis of certifying a child to receive WIC benefits. Most WIC programs that perform EP tests use them to screen for iron deficiency, although hematocrit or hemoglobin measurements are most commonly used for this purpose. The nutritional education and supplemental food provided by WIC are undoubtedly important in reducing lead absorption in many children and pregnant women.



Limited data on EP screening of children being seen for WIC certification or follow-up are available from CDC's Pediatric Nutrition Surveillance System (PedNSS). For calendar year 1989, 2,231,939 WIC visits for children 6 months through 4 years of age were reported to PedNSS (provisional data). Six States reported performing EP tests on 44,852 children; of these children, 10.8% had EP levels > 35 ug/dL. Data are not available on how many of the children with elevated EP levels had blood lead levels measured.

Head Start provides a comprehensive developmental program for low-income children between the ages of 3 and 5 years. About 24 percent of U.S. 3- and 4-year-olds living in poverty are served by 229 Head Start programs. Although Head Start is mainly known as an education program, 99 percent of the enrolled children receive medical screening (54 percent through EPSDT). This screening can include screening for lead poisoning, if lead poisoning is prevalent in the community. National data on how much lead screening is conducted through Head Start are not available.

In 1985-86, about 785,000 children were screened through childhood lead poisoning prevention programs (ATSDR, 1988). In Fiscal Year 1988, according to data collected by the Public Health Foundation, State and local health agencies screened 970,768 children and identified 18,912 that had positive screening tests requiring diagnostic confirmation (Jane Lin-Fu, personal communication). (These latter numbers include some children screened through EPSDT, W12, and Head Start, but they may underestimate the numbers of children screened under the MCH Block Grant Program.) Given that an estimated 250,000 children had blood lead levels above 25 ug/dL in 1984 (ATSDR, 1988), it is apparent that most lead-poisoned children are never identified.

REASONS TO INCREASE ACTIVITIES

- Increase the number of children screened
- Increase the use of intensive screening methods
- Ensure prompt investigations of the environments of poisoned children
- Assure proper follow-up of poisoned children



More childhood lead poisoning prevention activities are needed to 1) increase the number of children screened, particularly in communities with the highest levels of blood lead in children and rates of childhood lead poisoning, 2) increase the use of intensive screening methods, such as community outreach and door-to-door screening, 3) ensure prompt investigation of the environments of poisoned children, and 4) assure proper follow-up of poisoned children. Increasing the number of States that require or encourage EP or blood lead testing through MCH Bloc's Grant activities, EPSDT, WIC, and Head Start would probably be an efficient way of increasing screening in high-risk populations. Outreach and educational activities from the Federal level to regional and State offices and local agencies and programs could increase recognition of the importance of such screening. Better information about the amount and efficacy of screening children in EPSDT, WIC, and Head Start would be helpful in developing strategies for increasing testing through these programs where appropriate.

Other Efforts to Increase Screening and Education

OTHER EFFORTS NEEDED

- Increased outreach to children without a usual source of care
- Increased screening by health care providers
- Increased public awareness



Outreach to Children Without a Usual Source of Medical Care

On the basis of 1988 data from the National Health Interview Survey, it has been estimated that 8 percent of children less than 5 years of age do not have a regular source of medical care. Intensified childhood lead poisoning prevention activities must be directed at these children, many of whom are at high risk for lead poisoning. Some of these children could be reached by increasing enrollment in EPSDT and other programs. Others could be identified through intensified (for example, door-to-door) screening by childhood lead poisoning prevention programs. Additional strategies, such as screening children using emergency rooms in high-risk neighborhoods for primary or semiemergent care, should be evaluated for cost-effectiveness.

Screening by Health Care Providers

Education is of vital importance in increasing the amount of screening conducted by health-care providers. The American Academy of Pediatrics issued its most recent statement on lead poisoning prevention, diagnosis, and treatment in 1987. Nevertheless, many providers do not consider screening for childhood lead poisoning to be a part of routine pediatric care.

Several strategies are available for increasing health-care provider awareness. The first is to disseminate educational materials and do outreach through existing professional organizations and medical schools. (A partial list of relevant professional organizations is in Appendix IV, Table 1). A second strategy is to develop and disseminate training modules that can be completed for Continuing Medical Education credits, such as the Case Study in Environmental Medicine developed by ATSDR. A third is to provide conferences for medical care providers on childhood lead poisoning, either through the private sector (such as those held in 1989 at the University of Maryland and the University of Virginia) or through federally funded centers (such as the Health Education Centers of the Health Resources and Services Administration, the Education Resource Centers of the National Institute for Occupational Safety and Health, and the occupational and environmental clinics with activities funded by ATSDR).

Increased Public Awareness

Campaigns to increase public awareness of childhood lead poisoning and its prevention are likely to increase the amount of screening conducted. Such campaigns will not only educate medical care providers, they will also increase the public's demand for lead screening of children. Some lead poisoning cases may be prevented, for example, by informing homeowners of the potential dangers in renovating older homes. The National Maternal and Child Health Clearinghouse is a source of publications about childhood lead poisoning. This Clearinghouse and other resource centers could expand their activities, including operating a toll-free hotline and developing and disseminating simple materials about lead poisoning prevention in different languages. Information centers could also supply information on Federal, State, and local resources for dealing with childhood lead issues.



State and local health departments and childhood lead poisoning prevention programs should also be encouraged to increase public awareness of childhood lead poisoning. The categorical grants program authorized by the Lead Contamination Control Act of 1988 specifically allows funds to be used for educational activities conducted by State and local childhood lead poisoning prevention programs.

Several private sector organizations have sponsored educational activities about childhood lead poisoning. Other organizations should be encouraged to follow suit. Because childhood lead poisoning is associated with decreased intelligence and ability to learn, coalitions between organizations promoting lead poisoning prevention and organizations promoting education and the prevention of mental retardation should also be encouraged. Organizations that might be interested in such activities are listed in Appendix IV, Table 2.

Development of Infrastructure to Support Increased Childhood Lead Poisoning Prevention Programs

DEVELOPMENT OF INFRASTRUCTURE TO SUPPORT PROGRAMS MEANS INCREASED

- Training programs
- Laboratory services
- Laboratory proficiency testing programs



The expansion of screening programs will result in a demand for training programs on childhood lead screening and the investigation of environmental sources. The Louisville, Kentucky, training program can serve as a model for other such programs. This program provides methods for assessing lead poisoning in high-risk populations and demonstrates the integration of lead screening with basic child health services and the technical and management skills needed for an effective and efficient childhood lead poisoning prevention program.

In addition, increased screening will lead to a demand for increased laboratory services. In 1991 CDC will likely issue new recommendations suggesting that screening programs attempt to identify children with blood lead levels below 25 ug/dL. This change will mean that blood lead measurements must be used for childhood lead screening instead of EP measurements. When this happens, the demand for increased blood lead testing will far exceed current capacity. In addition, cheaper, easier to use, and portable instrumentation for blood lead testing will need to be developed. Furthermore, existing programs for proficiency testing and certification of laboratories will have to be expanded. With concern about health effects at low blood lead levels, laboratories will be called upon to do better measurements in the 4 to 5 ug/dL range. As a result, major efforts will be needed to improve laboratory quality assurance and control at these lower levels. Reference materials for laboratories performing blood lead measurements and technical assistance will be required to improve laboratory quality.



PROGRAM AGENDA ITEM 2. INCREASED ABATEMENT OF LEADED PAINT AND PAINT-CONTAMINATED DUST IN HOUSING

INCREASED ABATEMENT REQUIRES

- Setting priorities for which homes are to be abated first
- Strategies for increasing the number of abatements conducted
- Assuring the safety and effectiveness of abatement
- Development of infrastructure for abatement
- Development of a national implementation plan

Lead-based paint abatement is an integral part of the treatment of childhood lead poisoning and a crucial step in the prevention of new cases. Many sources besides lead-based paint are contributors to the exposure of children to lead, but we have four reasons for focusing on abatement of lead paint in this plan. First, lead-based paint and paint-contaminated house dust are still the major cause of high-dose lead poisoning in U.S. children. Second, we have known of the dangers of lead paint since the beginning of the century. The greatest concentrations of lead in paint occur in housing built before 1950. Although the Consumer Product Safety Commission has required paint manufactured for residential use to be almost lead-free since 1977, we have made little progress in eliminating paint previously applied as a cause of childhood lead poisoning. This problem may get worse with time, as houses painted with lead-based paint deteriorate further. Third, abatement of paint is expensive, and a successful effort to eliminate poisoning from leaded paint will require a coordinated effort from the



government and private sectors. Fourth, leaded paint abatement is difficult and potentially dangerous. Poorly performed abatements have poisoned workers and their families and people living in the homes being abated. In recent years, numerous families have been poisoned while renovating homes that were not tested for lead. Until this environmental source of lead is eliminated, the United States will continue to have a significant childhood lead poisoning problem.

Setting Priorities for Lead-Based Paint Abatement

PRIORITIES FOR ABATEMENT

- Homes of children identified with lead poisoning
- Homes at high risk of housing children with lead poisoning
- Homes with lead-based paint that are being renovated or remodelled for other reasons

An estimated 30 to 40 million residences in the United States contain leaded paint (ATSDR, 1988), although not all of them pose an imminent hazard. Priorities for abatement should be based largely on public health concerns; therefore, abatement programs must work in tandem with childhood lead poisoning prevention programs to ensure the most efficient use of resources.



Three priority groups of housing for abatement can be identified: homes of children identified with lead poisoning, homes at high risk of housing children with lead poisoning (but in which poisoned children have not yet been identified), and homes with lead-based paint that are being renovated or remodelled for other reasons. Although not specifically discussed in the following, day care centers and other buildings frequented by young children is also a high priority.

The first priority for abatement is the homes of children identified with lead poisoning. This is important not only to protect these children from continued exposure, but also to prevent children who will live in these dwellings in the future from being poisoned. In particular, children with lead poisoning severe enough to require medical intervention (i.e., ≥ 25 ug/dL) should be the utmost priority.

The second priority for abatement is the homes with a large potential for poisoning children. These are homes that are likely to be causing unrecognized lead poisoning or to poison children in the near future. This category includes housing in areas with a high prevalence of lead poisoning, but could include older housing in areas where there is little or no childhood lead screening. Screening, housing, socioeconomic, environmental, and other data should be used to identify those areas where housing is most likely to poison children. Abatement of housing in this category is a crucial part of the lead poisoning prevention strategy. Within this second priority group, decisions will have to be made about which specific homes and areas should be abated first. These decisions should be based on a combination of environmental and demographic data. A "hazard ranking scheme" should be developed and validated. The more efficient the identification of homes likely to contain poisoned children and to poison children in the future, the more cost-effective the abatement will be.

Opportunistic abatements, the third priority, involve those homes that can be efficiently abated because they are being worked on anyway or have other special characteristics. An example of opportunistic abatement is the removal of leaded paint from public housing during comprehensive modernization. The comprehensive modernization program is effective because 1) the Federal government has authority over the housing to be abated and 2) lead abatement adds only a relatively small amount to the cost of ongoing modernization activities.

Data from several evaluations show that abatement of lead-based paint decreases children's blood lead levels (Kennedy, 1978; Rosen, 1990; Copley, unpublished data; Amitai et al., unpublished data). The data from these studies indicate that even less than complete abatements reduce children's blood lead levels. In general, the most thorough abatements are believed to be the most effective in reducing blood lead levels and residual lead in the environment. Given the limited resources for abatement, however, a balance must be struck between doing the best possible abatements in fewer units and using reasonably good, less expensive methods in more units. The cost-effectiveness of alternative paint abatement methods should be evaluated, and the cost of abatement should be reduced through the development of new methods and materials and the establishment of a larger infrastructure for abatement.



An important issue is that some of the housing stock, particularly in the inner cities, is deteriorated past the point of rehabilitation or may be in neighborhoods that are so economically depressed that buildings rapidly deteriorate and are abandoned. Extremely deteriorated buildings in declining neighborhoods with large numbers of abandoned units are very likely to be abandoned or razed in the next 5 years. Requiring complete abatement in such situations would be futile and could lead to families being dislocated. In such circumstances, the efficacy of preventive maintenance--cleaning and partial abatement with frequent environmental and blood lead testing--should be determined, and its role should be defined. In addition, when low-income units are abated, safeguards will be required to ensure that they remain available as low-income housing.

Strategies for Increasing the Number of Abatements

STRATEGIES FOR INCREASING ABATEMENTS

- Incentives
- Demonstration programs
- Testing and disclosure requirements
- Education and public awareness

Increasing the number of abatements performed will require a mixture of public and private sector efforts. Housing can be divided into several different sectors--for example, public housing, public-assisted rental units, privately owned rental units, and owner-occupied homes. Different strategies will be required to increase abatements for different kinds of housing. These strategies include positive and negative incentives, demonstration programs, and the use of test and disclosure requirements.



Positive and negative incentive strategies confer a financial benefit or other advantage, or withdraw a financial benefit or advantage, to promote or discourage certain behaviors. Incentive programs can be used to encourage testing for lead-based paint or abatement of identified hazards. Demonstration area programs would set aside entire neighborhoods that would be abated to serve as a model to encourage abatement elsewhere.

Another possible strategy would require testing for lead levels in housing and the disclosure of the test results. (These results would be recorded, so that units undergoing multiple transactions would not be repeatedly tested.) Requiring the abatement of units with high lead levels could be an additional option. Testing and disclosure could be required for all housing units or it could center around transactional "trigger events," such as renovation or remodeling, renting, sale, or transfer.

Education and public awareness strategies are critical to the success of abatement programs. They are designed to inform the general public, the housing industry, and other relevant parties about preventing childhood lead poisoning and the role of lead-based paint. These strategies are designed to mobilize the community to act voluntarily to address the problem of leaded paint in housing. Public education and awareness will prompt the market to encourage abatement by placing a higher value on an abated house or rental unit than on a nonabated dwelling. Without increased awareness of the dangers of lead-painted housing, incentive strategies will be ignored, and regulatory approaches will be less acceptable to the public.

The President's budget for FY 1992 includes \$25 million for the HOME program which will be administered by the Department of Housing and Urban Development (HUD). This program will assist low- and moderate-income private residential property owners, abate lead-based paint, and will be directed to homeowners with young children in high-risk housing. This program could provide a knowledge base for evaluating the effects of abatement.

Federal, State, and local governments and the private sector have roles in many of these strategies; different groups are appropriate for implementing different strategies. How these strategies should be used to ensure the abatement of homes in the three priority groups and the roles of different levels and agencies of government and the private sector should be dealt with in an implementation plan.



Development of Infrastructure for Abatement (See Appendix V for More Details on the Material in this Section.)

INFRASTRUCTURE DEVELOPMENT MEANS

- Developing testing and abatement guidelines
- Developing worker training and certification programs
- Evaluating emerging abatement technology
- Developing laboratory accreditation programs
- Ensuring the availability of insurance for contractors
- Arranging relocations for residents during abatement
- Developing guidelines for disposal of abatement debris

Although enough is known to start an effective national abatement program, the capacity to undertake large-scale abatement does not currently exist. Regulations to ensure the safety of workers and occupants and the quality of the abatement work are limited. Very few inspectors, abatement contractors, or workers have been trained to perform the needed work properly. Both contractors and property owners can have difficulty getting insurance. These deficiencies in the infrastructure for abatement must be corrected as quickly as possible so that a national abatement program can be developed. This section briefly describes the steps that must be taken to increase the national capacity to do safe and effective abatements. More details on infrastructure development appear in Appendix V.



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Guidelines for testing for lead-based paint and performing safe and effective abatements are essential. In April 1990, HUD issued the first national set of comprehensive technical guidelines (the HUD Interim Guidelines) for lead paint testing and abatement. These guidelines were developed by a committee of government and nongovernment experts for public and Indian housing authorities. Since the guidelines were developed for housing that is to be extensively modified during modernization by the Federal Government, they should be modified for use by States, localities, and individuals in situations where funds are not as available, time is a critical factor, and the unit is not being gutted for other reasons.

The development of guidelines should be followed by the development of government-sanctioned model training programs for assuring the quality and consistency of worker training. As the amount of leaded paint abatement increases, market forces will meet the growing demand for training programs. Government involvement may be necessary, however, to control the quality of instruction and to assure the competence of trainees. In addition, mandatory requirements for the certification of contractors and their workers, testers, and inspectors should be established either by government or trade organizations.

Lead-based paint abatement will probably not evolve exclusively as a separate industry and skill specialty. It is an integral and inevitable part of a variety of existing building trades: painting, plastering, masonry, flooring, cabinetry, carpentry, electrical, plumbing, insulation, and door and window replacement. Some home renovation contractors will probably specialize in lead paint abatement. Thus, lead-based paint abatement should be integrated into the various building trades. Because abatement is a potentially hazardous activity, all workers involved in home renovation and repair should be familiar with the special safeguards and techniques required.

Another potential benefit of a national abatement program is increased employment. As persons with little training develop the skills needed for lead-based paint abatement, they will be likely to vacate jobs that do not require training. Because this abatement work will require a large work force, often in neighborhoods with high rates of unemployment, the training and employment of local persons will have local economic and social benefits.

Lead exposures of persons performing abatement and other workers, especially of pregnant women and of women and men who have or are planning to have children, should be reduced. At present, abatement workers are not covered by the Occupational Safety and Health Administration (OSHA) general industry standard regulating worker exposure to lead. Instead, they are covered under the safety and health standards for the construction industry, which regulate lead exposure far less strictly. A standard is needed that takes into account new data showing adverse effects of lead on adults at lead levels below the current OSHA general industry standard. Abatement workers and their families should be protected by medical monitoring and medical removal provisions, as are potentially lead-exposed workers in general industry.



During the past few years, private firms have developed a variety of new products to reduce the costs of lead-based paint abatement. Standards and performance criteria must be established to assure the effectiveness of new products. Standards for laboratories evaluating environmental samples should also be developed.

Other constraints to rapidly expanding lead-based paint abatement programs are the unavailability of liability insurance for contractors and building owners performing abatement, the lack of programs for quality assurance of lead-based paint and dust laboratory analysis, and the lack of suitable temporary housing for families whose homes are being abated. Another constraint is uncertainty about the proper disposal of abatement debris. When lead is removed from buildings, it is, in effect, being concentrated; if lead is to be kept from being dispersed in the environment, there must be rules and regulations for its safe disposal.

Development of a National Implementation Plan for Abatement

AN IMPLEMENTATION PLAN FOR ABATEMENT SHOULD FOCUS ON

- Increased abatements by the private and public sectors
- Increased safety and efficacy and decreased cost of abatement
- Targeting of high-risk housing
- Best use of available funds

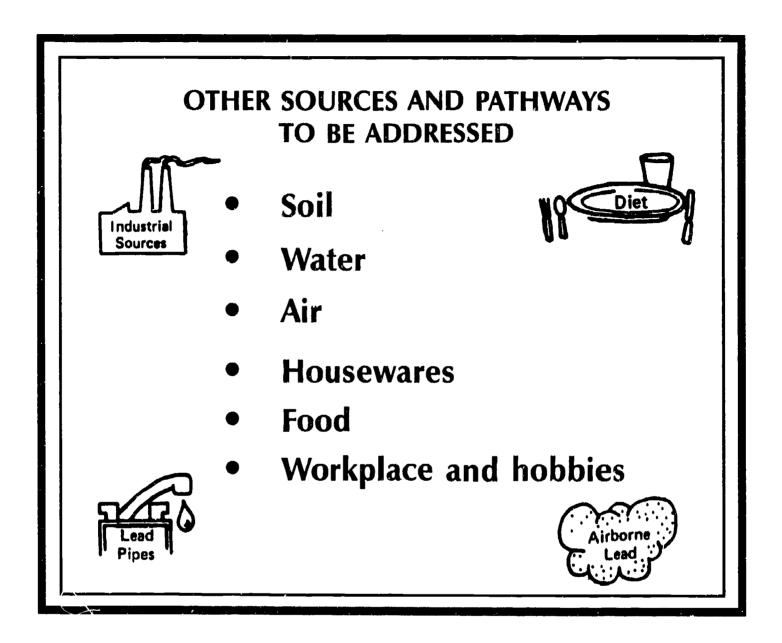


A well-designed national implementation plan for increasing the number of abatements performed should be developed immediately. Although there is an immediate need for increased resources for abatement, a phased approach to increasing abatement should be designed. The implementation plan should focus on three main issues: 1) how to increase private and public sector abatements; 2) how to increase the safety and efficacy and decrease the costs of abatements through technology development and evaluation and worker training and certification; and 3) how best to use available funds to quickly reduce the number of children poisoned by lead-contaminated housing.

During the early years of the national abatement strategy, an evaluation component will be essential. This evaluation should include measurements of efficacy and safety through postabatement environmental and human testing, and the inspection and collection of data on numbers of abatements being funded by the private and public sectors.



PROGRAM AGENDA ITEM 3. REDUCTIONS IN O'HER SOURCES AND PATHWAYS OF LEAD EXPOSURE



Lead-based paint and paint-contaminated dust account for most cases of lead poisoning in the United States. Other sources of lead will also have to be addressed, however, to eliminate this disease. For example, lead-contaminated soil is probably an important source for a large number of children. However, adequate information is not yet available on which to base recommendations for a national soil abatement strategy. Federal agencies are proceeding with or are evaluating further regulation of environmental lead in water, air, and housewares. In this section, some current and needed activities are summarized.



Although lead-based paint and paint-contaminated dust account for most cases of childhood lead poisoning in the United States, other sources of lead will also have to be addressed.

The Environmental Protection Agency (EPA) is evaluating the need for more stringent standards for lead in drinking water and air. EPA is also conducting a demonstration project in three cities to evaluate the benefits of removing lead-contaminated soil from yards of homes where children live.

The Food and Drug Administration (FDA) has proposed new regulatory standards for lead in ceramic pitchers and other types of ceramic foodware. FDA is also attempting to identify sources of lead in the diet other than those that have already been identified, such as lead in wine bottle cap wrappers and in calcium supplements. Mechanisms should be established so that potters and other crafts people either clearly indicate that their wares are not for food service or have their wares tested to ensure that they do not contain lead.

In coordination with FDA, domestic manufacturers of food cans have markedly reduced their use of solder with a high lead content. This change has resulted in large reductions in the lead levels in canned foods in the United States. Nevertheless, a total ban on the use of solder with high lead content in domestically produced canned goods should be seriously considered. The frequency of use of solder with high lead content in imported food cans is unknown; a ban on the use of solder with high lead content in imported food cans should also be considered.

Childhood exposures from parental occupations and hobbies involving lead should be reduced. This can be done through a combination of good work practices and education. The use of folk remedies containing lead continues to be a problem in certain ethnic populations. Educational activities, intensified lead screening, and intervention strategies could reduce exposure to this source of lead.



PROGRAM AGENDA ITEM 4. NATIONAL SURVEILLANCE FOR ELEVATED LEAD LEVELS

USES OF SURVEILLANCE DATA

- To target interventions
- To track progress
- To evaluate worker exposures

The only national data available for estimating the number of children who may have elevated blood lead levels are derived from national surveys of nutritional and health status that, in the past, have been conducted about once a decade. These data are extremely valuable for providing unbiased estimates of the blood lead levels of children and workers in the United States. In the future they will be conducted more often, and this will make it possible to evaluate national and regional blood lead levels more frequently. As these data are now collected, however, they cannot be used to monitor short-term trends over several months or a few years. They cannot be used to characterize geographic distributions of poisoning in the community or to target interventions where they are most needed. A national surveillance program for elevated blood lead levels in children and workers is essential for the development of a "lead priority list" for targeting interventions, for tracking our progress in eliminating childhood



lead poisoning, and for evaluating lead exposure in abatement workers and workers in other lead-contaminated environments.

Several sources of data could be used for surveillance. These include childhood lead poisoning prevention programs, other government programs that conduct or reimburse for screening for lead poisoning, and laboratories that perform blood lead testing.

The development of better systems for managing data in childhood lead poisoning prevention programs should be a high priority. Data from childhood lead poisoning prevention programs could be extremely important for evaluating the yield of screening in specific areas, the yield of alternative screening strategies, and the efficacy of interventions. Since screening takes place in only limited geographic areas, however, data from screening programs cannot provide national information. Furthermore, although many areas that need targeted abatement programs could be identified through screening data, areas that have no screening programs could not be evaluated. In addition, many large programs have not yet computerized their data, and those computer systems that exist are often cumbersome or cannot link data on screening and medical follow-up with data on environmental investigations and interventions.

Data from other go ernment programs conducting or reimbursing for screening, like EPSDT, could also be useful, but these data have serious limitations. They would provide information on only a small segment of the population being tested for lead poisoning, and they would not include follow-up data.

The optimal model for national surveillance is the notifiable disease system that CDC has used lince 1961. Through this system, cases of illnesses are reported electronically to CDC by State epidemiologists. Since lead poisoning is diagnosed on the basis of laboratory tests, reporting for lead would depend upon laboratories sending their data on persons with elevated blood lead levels to State health departments for transmission to CDC. The State health department would also be responsible for ensuring that multiple tests on the same individual are identified as such and that persons needing follow-up are referred appropriately. An evaluation component is essential for determining that the data collected are complete and representative. The American Academy of Pediatrics; the American Medical Association, and the Council of State and Territorial Epidemiologists have endorsed the development of such surveillance.



KEY ORGANIZATIONS ENDORSING NATIONAL SURVEILLANCE

- American Academy of Pediatrics
- American Medical Association
- Council of State and Territorial Epidemiologists

The feasibility of developing national surveillance for elevated lead levels is illustrated by the National Institute for Occupational Safety and Health (NIOSH) efforts to develop a system for reporting elevated blood lead levels in workers. NIOSH receives reports from eight State health departments that provide data about numbers of workers with elevated blood lead levels and industries in which lead poisoning is occurring. The States with surveillance systems also ensure follow-up of the affected workers. In 1988, 4,804 workers in seven States were reported to have blood lead levels ≥ 25 ug/dL.



CHAPTER 5. RESEARCH AGENDA

RESEARCH IS NEEDED FOR

- INCREASED PREVENTION ACTIVITIES
- INCREASED ABATEMENTS
- REDUCTIONS IN OTHER SOURCES

Enough is already known to start an effective campaign to eliminate childhood lead poisoning, and intensified efforts to prevent this disease should get under way immediately. There are, however, several questions that must be answered if this disease is to be successfully eradicated in the most cost-effective manner. The following are key elements of a research agenda designed to provide essential information for future years of a program to eliminate childhood lead poisoning. Many of these elements appeared in the Committee to Coordinate Environmental Health and Related Programs ad hoc committee report of the implementation of the ATSDR report to Congress.



The results of basic research have shown the need for a strategic plan for the elimination of childhood lead poisoning.

This research agenda does not include a discussion of or a budget for many basic research activities. Such activities include evaluating the amount of lead absorbed by children and adults, identifying new biomarkers for lead exposure, and determining the impact of pharmacological treatment of lead poisoning on children's cognitive functioning. Although these activities are not essential for the first 5 years of the Strategic Plan, they are important. The findings of basic research have made a plan such as this necessary, and they make it possible to develop a program agenda at this time. These research activities should receive financial support.

RESEARCH AGENDA ITEM 1. RESEARCH FOR CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES

RESEARCH FOR INCREASED CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES

- Cost-effectiveness of screening strategies
- Better instruments for blood lead testing
- Evaluation of capillary blood collection devices
- Evaluation of educational and nutritional interventions



Studies should be conducted on the cost-effectiveness of different strategies for childhood lead screening. These strategies include screening in inner-city emergency rooms to reach children who have no ongoing source of care and "cluster testing" of all children in multiple dwelling units where cases of childhood lead poisoning have been identified. The usefulness of screening in day care centers and nursery schools should also be evaluated. In addition, Federal programs now funding childhood lead screening should be evaluated to see how they can work together for a most efficient use of resources.

At present it is much cheaper and easier to perform an EP test than a blood lead measurement; however, the EP test is not a useful screening test for blood lead levels below 25 ug/dL. Both because of the expected increase in screening and because of the concern about the health effects of lower blood lead levels, the demand for blood lead testing is likely to increase. The development of portable, easy-to-use, cheaper instrumentation for blood lead measurement is extremely important.

Because capillary (or fingerstick) blood samples may be easily contaminated with lead on the skin, venous blood must be used to confirm lead poisoning in children. Several capillary blood collection devices now on the market purport to collect blood free of surface finger contamination from lead. These devices should be evaluated for ease of use and ability to collect an uncontaminated sample.

The education of families about lead poisoning by childhood lead poisoning prevention programs often includes information about the importance of nutrition. Because of our growing concern about the adverse effects of low blood lead levels, nutritional interventions are likely to be recommended for more children. A number of nutritional factors have been shown experimentally to influence the absorption of lead and its concentrations in tissues. Intervention studies or clinical trials should be conducted to establish that increasing the regularity of meals and ensuring adequate dietary intake of iron and calcium can reduce blood lead levels.

Educational strategies for increasing medical care provider and public awareness of lead poisoning should also be evaluated for their efficacy in reducing children's blood lead levels and preventing lead poisoning.



RESEARCH ON ABATEMENT

- Long-term follow-up postabatement
- Efficacy of abatement methods
- Better methods for measuring lead in paint and dust
- Evaluation of worker exposures
- Abatement of forced air ducts, rugs, furniture, etc.
- Determination of safe environmental levels
- Efficacy of preventive maintenance

The techniques recommended in the HUD Interim Guidelines have been shown to be effective in abating lead-based paint and reducing dust levels. However, no long term evaluations have been conducted to ensure that dust lead levels and children's blood lead levels remain low once abated units have been reoccupied. Long-term follow-up of units abated under these guidelines and their occupants should be conducted.

Few childhood lead poisoning prevention programs perform as rigorous an abatement as that recommended in the HUD Interim Guidelines. Better data on the long-term efficacy of less stringent abatement methods should also be collected, and the cost-effectiveness of alternative methods of lead-based paint abatement should be evaluated. These analyses should be used to determine how best to spend resources,



given that more complete and expensive abatements probably result in greater reductions of blood lead levels but may result in fewer units being abated.

Current methods for measuring lead in paint and dust are sometimes inaccurate, expensive, or both. Accurate, inexpensive methods for such measurements would decrease the cost and increase the reliability of preabatement and postabatemement testing. These methods include improved X-ray fluorescence (XRF) devices and chemical spot tests. Preferred methods are those that can be used onsite, instead of requiring offsite laboratory analysis, and those that do not destroy surfaces.

All abatement methods should be evaluated to determine worker exposures to lead and other hazards. Laboratory and field studies should be conducted, when appropriate, before new methods are recommended for widespread use, and they should include evaluations of worker safety. HUD, EPA, and other agencies have already started some of these evaluations.

Methods for abating such items as forced air ducts, rugs, and furniture have not been evaluated adequately. Furthermore, there is no consensus on whether such abatement is appropriate. For example, discarding lead-contaminated rugs and upholstered furniture has been advocated.

Environmental lead levels used for determining whether a home needs abatement or if an abated unit can be reoccupied are based on limited scientific data. These levels should be evaluated to ensure that they are both adequate to protect health and do not result in unnecessary abatements. Included in this work would be the paint lead concentration at which paint abatement is recommended; the dust lead concentration at which dust abatement is recommended, even in the absence of lead-based paint; and the soil lead concentration at which abatement should occur. In addition, a system for estimating the total lead hazard in a building or housing unit, combining information on household demographics, paint lead concentration, quantity, and condition, and dust and soil lead levels should be developed. The lead levels of paint, dust, and soil that are to be considered safe after abatement should be evaluated. One important outcome of this work would be algorithms for identifying which housing units are most likely to poison children in the future.

Because of the limited money available for abatement, inexpensive interim methods must be developed and evaluated for preventing children from being exposed to high environmental lead levels in units awaiting abatement. Such interventions may include regular professional cleaning with high-efficiency vacuum cleaners and scraping and repainting small areas of peeling paint. Outcome measurements should include measurement of lead in house dust and children's blood. Such preventive maintenance strategies should be evaluated over several years.



RESEARCH ON REDUCTIONS IN OTHER SOURCES AND PATHWAYS

- Relative contributions of different sources
- Cost-effectiveness of soil abatement
- Drinking water lead levels and treatments
- Sources of dietary lead
- Improved food lead measurement



- Bioavailability
- Mobilization of lead during pregnancy

Studies should be conducted to determine the relative contributions of various sources and pathways of lead to children's blood lead levels. These studies should also investigate the relationship between lead in the various environmental compartments to which children are exposed. Sources and pathways to be investigated should include paint, dust, soil, air, food, water, and exposure from parental occupations and hobbies.

Current methods for remediating soil are expensive, and their efficacy under varying conditions has not been proven, particularly in urban areas. Studies should be conducted to examine the efficacy and cost-effectiveness, in terms of blood lead level reductions, of various methods of remedia , soil (such as removing soil and planting ground cover). These efforts should complainent EPA's ongoing efforts.



Lead levels in drinking water in the United States, including levels in water fountains, should be assessed more completely. Alternative treatment approaches aimed at reducing lead in drinking water should be evaluated.

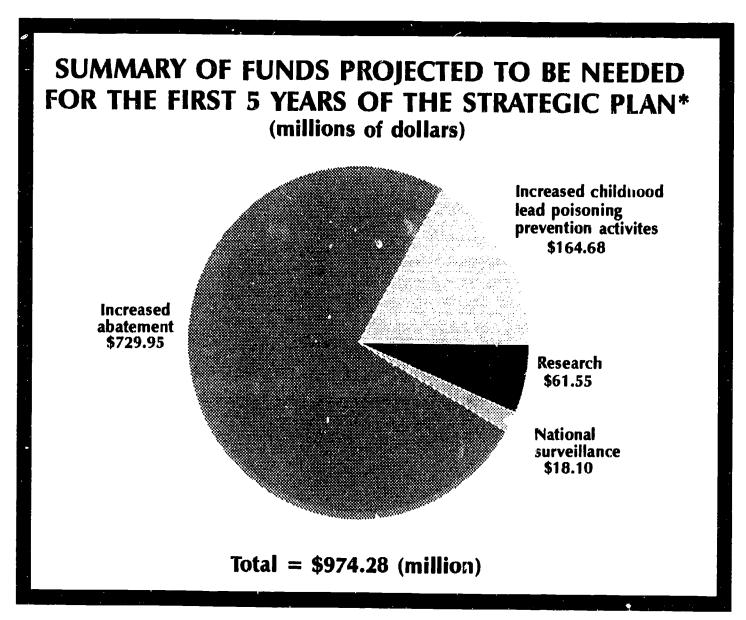
While a great deal is known about many dietary sources of lead, others have not been identified or evaluated. Lead in calcium supplements is of particular concern because of the many pregnant women taking these preparations. Other inadequately studied sources of lead include wine (from lead in the wine itself or in caps or seals), coffee produced in institutional coffee urns, infant foods, and bottled waters. Surveys of lead ingested by special populations should also be conducted. These surveys should focus on canned foods, housewares, and folk remedies used by special populations, such as ethnic groups. For these evaluations, analytical procedures will have to be improved.

The bioavailability of lead probably varies according to the substrate (for example, paint, dust, soil, food) and the chemical form and particle size of the lead. Criteria for cleanup may need to vary according to the probable bioavailability of lead at a given site. Animal feeding studies and collection of data on human populations are needed to provide information on how bioavailability issues should be considered when decisions on remediation and clearance are made.

Studies should be conducted on the mobilization of bone stores of lead during pregnancy and on the biokinetics of fetal lead exposure. If bone stores prove to be an important determinant of blood lead levels during pregnancy, interventions to reduce lead mobilization in pregnancy should be developed and studied.



CHAPTER 6. FUNDS NEEDED FOR IMPLEMENTATION OF THE STRATEGIC PLAN



*Costs reflect the amount of money needed to implement the program agenda and a shared commitment of the public and private sectors.

Our estimate of the cost of implementation for the first 5 years of the Strategic Plan is expected to be \$974 million. Ninety-four percent of this money is for program activities; six percent is for research. The source of funds is not discussed in this report; these costs reflect a shared commitment of the public and private sectors.



Funds Needed for Implementation of the Program Agenda

Implementation of the program agenda will require the efforts and cooperation of many Federal, State, and local agencies. The first five years of this agenda will cost \$913 million. This budget does not include funds for program activities needed to reduce sources and pathways of exposure other than lead-based paint and paint-contaminated dust. Many of these are already being addressed through Federal and other actions.

The estimate of the additional costs for increased abatement requires further discussion. Because of the lack of baseline data, it is difficult to project now many more housing units should be abated as part of a strategic plan to eliminate childhood lead poisoning. Furthermore, development of cheaper abatement methods and of an infrastructure for abatement is an essential part of the first years of any national abatement strategy. Therefore, a phased increase in the number of abatements performed is proposed, with an emphasis on research and development and the testing of strategies and materials in the first 2 years of the program. Within 3 years, resources should be made available to perform 20,000 to 30,000 more abatements annually than are currently being performed. These resources would be enough to abate the homes of all lead-poisoned children currently being identified by childhood lead poisoning prevention programs who have no other source of funding for abatement. (As the amount of screening increases, the estimate of additional units to be abated annually will also need to be increased.) These resources would also make it possible to have demonstration projects and to abate units in the second priority group, homes that have a large potential for poisoning children. At this rate, eliminating all lead paint from housing stock in the United States will take a long time, but it is important to make a start--to eliminate lead-based paint from those units that have the greatest potential to adversely affect health.

The costs of abatement vary greatly according to the size and kind of housing unit, the region of the country, and other factors. For this plan, we assumed that an average abatement costs around \$6,500. This estimate was developed by Anne Elixhauser, Battelle, under a contract with CDC through interviews with screening programs. (The abatement methods used in the three studies whose data form the basis of the benefits analysis and the cost-benefits analysis in Appendix II were much cheaper and less comprehensive; however, data are not available on how much blood lead levels might be reduced by more expensive methods. We assume that the reduction would be correspondingly greater. Information on the costs and benefits of abatement will need to be continually updated as new information becomes available.) Thus, the abatement of 20,000 to 30,000 units a year could be expected to cost around \$130 to \$195 million a year. Since it will take a couple of years to build up the infrastructure for abatement and increase the number of abatements performed, we estimate that the increased abatements needed to complete the first 5 years of this Strategic Plan would cost a total of \$710 million. The unit cost of abatement is likely to decrease over the next several years as new abatement methods are developed and the infrastructure for abatement increases.



Increasing the amount of abatement conducted will also require development of a national abatement plan and infrastructure development, as described in Chapter 4. We estimate that the costs of such development work will be between \$3 and 6 million a year for the first 5 years of the Strategic Plan, and will total \$19.95 million over 5 years.

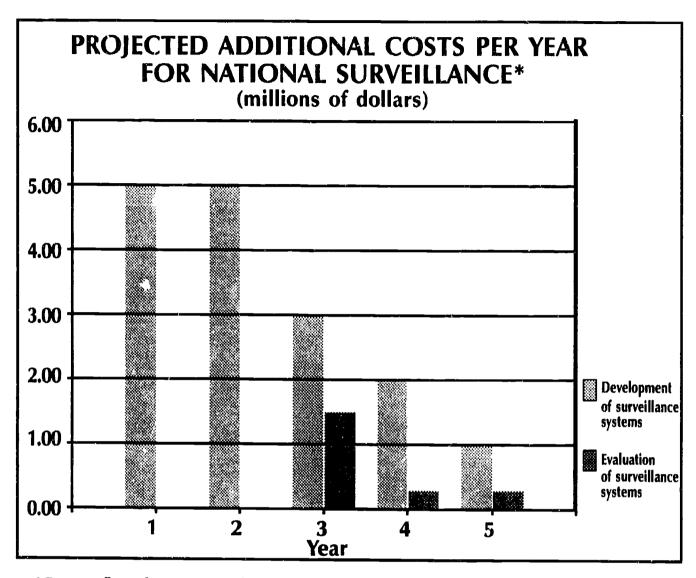
Following are detailed budgets for increased childhood lead poisoning prevention activities and national surveillance.

PROJECTED ADDITIONAL COSTS PER YEAR FOR INCREASED CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES*

		Cost per Year (millions of dollars)					Tota Cost
	Year	1	2	3	4	5	
Increased funding for programs		25	25	25	35	45	155
Increased screening through EPSDT, WIC, and Head Start		0.25	0.25	0.25	0.25	0.25	1.25
Educational materials and outreach		0.5	0.3	0.01	0.01	0.01	0.83
Federal campaign to increase awareness		0.5	0.5	0.5	0.5	0.5	2.5
Clearinghouse		0.75	0.5	0.2	0.1	0.05	1.6
Infrastructure development for prevention activities		1.5	0.8	0.8	0.2	0.2	3.5
Total		28.50	27.35	26.76	36.06	46.01	164.68

^{*}Costs reflect the amount of money needed to implement the program agenda and a shared commitment of the public and private sectors.





*Costs reflect the amount of money needed to implement the program agenda and a shared commitment of the public and private sectors.



Funds Needed for Implementation of the Research Agenda

Implementation of the research agenda will cost \$62 million. The next three tables summarize the budgets for the three main categories of research needed to support the program agenda.

PROJECTED ADDITIONAL COSTS PER YEAR FOR RESEARCH FOR CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES*

		Cost per Year (millions of dollars)					
	Year	1	2	3	4	5	
Cost-effectiveness of alternative screening strategies		0.5	0.5	0.5	-	•	1.5
Capillary collection devices		0.3	0.1	-	-	•	0.4
New instrumentation for measuring blood lead levels		0.2	0	-	**	-	0.2
Nutritional interventions		2	1	1	0.25	-	4.25
Educational strategies		2	1	0.5	0	0	3.5
Total		5.0	2.6	2.0	0.25	0	9.85

^{*}Costs reflect the amount of money needed to implement the research agenda and a shared commitment of the public and private sectors.



PROJECTED ADDITIONAL COSTS PER YEAR FOR RESEARCH ON ABATEMENT*

			Total Cost				
	Year	1	2	3	4	5_	
Efficacy of abatement		1.3	0.5	0.5	0.5	0.5	3.3
Alternative abatement methods		2	2	2	1	0.5	7.5
Measurement of lead in paint and dust		0.6	0.1				0.7
Worker exposure studies		0.5	0.5	0.2	0.2	0.2	1.6
Abatement of air ducts, etc.		1			~~		1
Safe levels of lead in paint, dust, and soil		1	2	2	1	1	7
Preventive maintenance		8.0	0.5	0.2	0.2	0.2	1.9
Total		7.2	5.6	4.9	2.9	2.4	23.0

^{*}Costs reflect the amount of money needed to implement the research agenda and a shared commitment of the public and private sectors.



PROJECTED ADDITIONAL COSTS PER YEAR FOR RESEARCH ON REDUCTIONS IN OTHER SOURCES

	Cost per Year (millions of dollars)					Total Cost
Year	1	2	3	4	5	
Sources of children's exposure	1	1.5	1.5	0.5	0.5	5
Cost-effectiveness of soil abatement	2	2	2	2	1	9
Drinking water lead levels	1	0.5				1.5
Treatment for lead in water	0.3	0.3	••			0.6
Sources of dietary lead	2	1	1	1	0.6	5.6
Food lead measurement	0.4	0.15	0.15	0.15	0.15	1
Bioavailability studies	1	1				2
Lead biokinetics in pregnancy	1	1	1	0.5	0.5	4
Total	8.7	7.45	5.65	4.15	2.75	28.

^{*}Costs reflect the amount of money needed to implement the research agenda and a shared commitment of the public and private sectors.

CHAPTER 7. SUMMARY OF RECOMMENDATIONS

In summary, childhood lead poisoning is a preventable disease with a huge societal cost. This plan outlines several steps that must be taken to eliminate sources of lead exposure for children. These steps will require a combination of government financial assistance and strategies to maximize the role played by the private sector.

The most urgent elements of the plan are the following:

- o Increased childhood lead poisoning prevention activities -- These activities are essential to identify poisoned children and assure appropriate interventions are conducted. They are also important for targeting neighborhoods that need more intensive, communitywide interventions for preventing lead poisoning.
- o Increased abatement -- A nationwide lead-based paint abatement program must be designed that will maximize the number of children benefited, given the fixed resources for abatement, using safe and effective methods.
- o Reductions in other sources and pathways--Ongoing efforts to limit children's exposure to lead from water, food, air, soil, and the workplace require continued attention.
- o Surveillance -- A national surveillance system for elevated blood lead levels should be developed for tracking progress in eliminating childhood lead poisoning, identifying areas in need of further evaluation or interventions, and evaluating exposures of persons performing abatement and other workers.
- Research Research on lead should focus on developing and evaluating cost-effective methods for screening children, testing paint and dust for lead, and reducing the sources of lead to which children can be exposed as much as possible.



REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). The nature and extent of lead poisoning in children in the United States: a report to Congress. Atlanta: U.S. Department of Health and Human Services, 1988.

Bellinger D, Sloman J, Leviton A, Rabinowitz M, Needleman H, Waternaux C. Low-level exposure and children's cognitive function in the preschool years. Pediatrics 1991;57:219-227.

Centers for Disease Control (CDC). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: U.S. Department of Health and Human Services, 1985; CDC report no. 99-2230.

Environmental Protection Agency (EPA). Air quality criteria for lead. Research Triangle Park, N.C.: Office of Health and Environmental Assessment, 1986; EPA report no. EPA/600/8-83/028aF.

Kennedy FD. The childhood lead poisoning prevention program: an evaluation. A report for the Centers for Disease Control. 1978.

Levin R. Reducing lead in drinking water: a benefit analysis. Washington, DC: Environmental Protection Agency. 1986; EPA report no. 230-09-86-019.

Needleman HL, Schell A, Bellinger D, et al. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. N Engl J Med 1990;322:83-8.

sen JF, Markowitz ME, Bijur PE, et al. Sequential measurements of bone lead content by L-X-ray fluorescence in Ca₂ EDTA treated lead-toxic children. Environmental Health Perspect (in press).



APPENDIX I

LEAD EXPOSURE AND ITS EFFECTS ON CHILDREN AND FETUSES

The problem of human exposure to lead has been extensively studied, probably more than exposure to any other toxic substance. For public health policy, the following summary findings are especially important:

- o Lead is an extremely dangerous and pervasive environmental poison.
- Today, far too many children are still exposed to excessive levels of lead: most recent national estimates indicate that in 1984, between 3 and 4 million children had lead in their bodies at levels which justify significant public health concern and which have been associated with neurobehavioral and other adverse health effects.
- Our children have not been effectively protected from the major sources of lead exposure--especially leaded paint and lead-contaminated dust and soil.

LEAD EXPOSURE

Lead has some unusual characteristics that cause special concern. First, lead deposited in the environment remains there and accumulates. Therefore, lead distributed in the areas where we live from paint, gasoline, and stationary sources remains there. As long as lead continues to be added to our environment, more lead will accumulate.

Second, lead exposure is pervasive, sparing no socioeconomic segment of the United States. Since lead is dispersed into air, food, soil, dust, and water, children of all socioeconomic backgrounds in all geographic areas experience unacceptably high lead exposures. Overall, children living in or around old, dilapidated inner city housing are at highest risk for lead poisoning.

Third, lead accumulates over months and years in the bodies of children. Therefore, chronic exposure to small amounts of lead can lead to a large long-term accumulation in a child, increasing that child's risk of adverse health effects. In addition, it is believed that, during pregnancy, women's body lead stores may be mobilized, exposing the fetus to lead. Therefore, childhood exposures in one generation may result in prenatal exposure for the next generation.

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Measurement of Lead Exposure

All Americans are exposed to some amount of lead. The amount of exposure has most often been quantified by measuring lead in blood (common units are ug/dL). Lead in blood reflects exposure during the previous weeks or months, whereas bone (or tooth) lead is a measure of cumulative lead exposure over months and years.

In most studies of the health effects of lead, measurements of outcome (such as IQ or behavioral changes) have been compared with blood lead measurements, and most public health decisions have been based on blood lead levels. Developing more practical methods to measure bone lead may substantially increase the use of such methods in assessing lead exposure; but at present, blood lead measurements remain the most generally used method of assessing human exposure to lead. The current definition of childhood lead poisoning is a blood lead level ≥25 ug/dL with an erythrocyte protoporphyrin (EP) level ≥35 ug/dL (Centers for Disease Control, 1985). This definition is currently being reevaluated, and the blood lead level will be revised downward to the level of 10-15 ug/dL.

Sources and Pathways of Lead Exposure

Children are exposed to lead from multiple sources such as paint, gasoline, solder, batteries, and stationary sources via multiple pathways such as air, dust, dirt, water, and food. The distinction between sources and pathways is not always clear. For example, dust and dirt are pathways for lead exposure. Because so much lead has been deposited in dust and dirt, they are sometimes also considered sources of lead exposure. In addition, in some discussions of lead exposure, water, food, and air are classified as sources of lead, although lead in these media comes almost totally from other sources.

The important public health point is that lead comes from known sources and moves through and is deposited in identified pathways to enter children. Although accurately tracing lead through all the complex pathways once it has left the source (e.g. leaded paint on a wall) may be difficult, it is not difficult to establish that reducing the amount of lead coming from the source will reduce the amount of lead going into children. For example, it is difficult to accurately trace all the pathways by which lead from gasoline enters children. Nonetheless, children's blood lead levels are well-correlated with gasoline usage patterns, and these levels have fallen dramatically in response to the reduction of lead in gasoline.

Children are exposed, therefore, when lead moves from its source through environmental pathways to be ingested or inhaled by a child. Reducing the amount of lead coming from these primary sources of lead (e.g. leaded paint on a wall) v.ill reduce children's exposure to lead.

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For the fetus, exposure comes from the mother's blood lead burden. The placental barrier is not effective in stopping lead from crossing over to the fetus (ATSDR, 1988). Generally, prenatal exposure is assessed by measuring the mother's blood lead level. The role of mobilization of maternal bone lead stores in prenatal exposure is yet to be determined.

For an individual child, the particular environment in which the child lives determines the relative importance of each lead source. For example, for a child living in a home with deteriorating lead paint, the paint will almost certainly account for a significant portion of exposure.

Although the immediate environment determines the importance of various lead sources for an individual child, estimates can be made of the overall relative importance of lead sources to U.S. children as a group. The Agency for Toxic Substances and Disease Registry (ATSDR) recently reviewed the available information on childhood lead exposure by source. The following are ATSDR's estimates of the number of children exposed by lead source (ATSDR, 1988). As noted in that report, these estimates are based on the best available information, and the estimation errors are difficult to quantify. The assumptions involved in the calculations differ for each source. Some numbers are for children potentially exposed and some for children actually exposed.

Lead in paint: Currently, leaded paint is the source of greatest public health concern. It is the most common cause of high-dose lead exposure. Exposure occurs not only when children ingest chips and flakes of paint (which often contain as much as 50% lead by weight) but also when children ingest lead paint-contaminated dust and soil, usually during normal mouthing activities. ATSDR has assessed that existing leaded paint in U.S. housing and public buildings is "an untouched and enormously serious problem."

About 13.6 million children under 7 years of age are potentially exposed in their homes to paint that contains lead at concentrations of 0.7 mg/cm² or higher. About 1.8 to 2.0 million children live in housing with unsound lead-based paint (e.g., holes in walls, peeling paint), which places them at high risk of excessive lead exposure; about 1.2 million of these children are estimated to have blood lead levels above 15 ug/dL, mainly due to exposure to leaded paint.

Lead-based paint abatement has been an essential part of all lead poisoning prevention programs in high-risk areas, despite cost constraints which limit the extent of such abatements. Historically, many studies have shown that the risk of lead poisoning is related to the presence of lead-based paint, and also to deteriorated or dilapidated housing (Gilbert et al., 1979); lead in dust is undoubtedly an important pathway for such exposures. Bornschein et al. and Chisholm have shown that children living in or returning to rehabilitated lead-free or lead-reduced housing after medical treatment for lead poisoning have significantly lower lead levels than children living in similar, non-rehabilitated housing (Bornschein et al., 1986; Chisholm, 1988). Three studies cited



in Appendix II demonstrate decreased blood lead levels in children with lead poisoning after the abatement of lead-based paint in their homes. (There may also be additional input to dust lead from lead in outdoor soil. Exposure to soil lead may occur from direct exposure to soil or indirectly as a result of its contribution to dust lead indoors. Lead in soil may arise from past use of exterior lead-based paint or from other external sources (see below). The value and role of soil abatement in addition to lead-based paint and dust abatement are currently being investigated in an Environmental Protection Agency (EPA) demonstration project; this issue will probably not be clarified for at least several years.)

Lead in gasoline: Since the introduction of lead as a gasoline additive in the mid-1920s, millions of tons have been used for this purpose (EPA, 1986). The recent reduction in the amount of lead in gasoline in the United States has been of major benefit to children. In the 13 years between 1976 and 1989, the amount of lead used in gasoline was reduced by more than 99% (EPA, 1990). Because of this, the blood lead levels in the U.S. population have decreased substantially.

Lead from primary and secondary smelters: About 230,000 children live near enough to a primary or secondary smelter to be exposed to lead from that source. Up to 13,000 of these children are estimated to have blood lead levels above 20 ug/dL from exposure to smelting by-products.

Lead in drinking water: In the United States, lead in water comes predominantly from lead in plumbing such as lead-soldered joints in copper pipes. The EPA maximum contaminant level (MCL) for lead is currently 50 ug/L. EPA estimated that in 1988, about 3.8 million children were exposed to water with a lead concentration higher than 20 ug/L. In 1988, EPA proposed that the allowable level for lead in drinking water be reduced. A revised lead standard is currently under consideration.

Lead in food: EPA estimated that about 42% of lead in food comes from lead-soldered cans or other metal sources, about 45% is deposited from the atmosphere, and the remainder comes from unidentified sources (EPA, 1986). Thus, almost 90% of lead in food comes from sources external to the food. Because of major decreases in the production of lead-soldered food and beverage cans and decreases in air lead levels due to decreases in gasoline lead, food lead levels are declining. The most obvious means of reducing lead in food is to reduce further lead in soldered cans and reduce lead emissions into the air (this has essentially been accomplished for mobile sources, i.e. automobiles, but not yet for stationary sources, such as smelters, incinerators, and other industrial sources).

Lead in dust and soil: Dust and soil act as a pathway to children for lead deposited by primary lead sources such as leaded paint, leaded gasoline, and stationary lead emitters. Since lead does not dissipate, biodegrade, or decay, the lead deposited into dust and soil becomes a long-term source of lead exposure for children. For example, although lead



emissions from gasoline are much reduced, gasoline lead deposited in years past remains in the dust and soil, and children continue to be exposed to it. The same is true for lead-based paint used in previous years. ATSDR (1988) has concluded that the "actual number of children exposed to lead in dust and soil at concentrations adequate to elevate blood lead levels cannot be estimated with the data now available."

Other sources and pathways of lead exposure: Several other sources and pathways are also important causes of elevated lead levels in many populations. These include lead in ceramic ware, folk remedies, hobbies or craftware, and childhood exposure to lead brought home by parents from their workplaces. As battery recycling increases, exposure to lead from this activity should be limited by control of emissions and lead levels in the workplace.

ADVERSE EFFECTS OF LEAD EXPOSURE

The adverse health effects on children from exposure to lead are a major public health concern. The risks associated with many chemicals, especially carcinogens, are extremely uncertain; for such chemicals, conservative approaches are used to extrapolate risks from animal or human occupational studies to estimate the upper limit of the risk posed to children and other populations. The adverse effects and risks of lead are well-known from studies of children themselves, and risk assessment calculations, with their inherent uncertainties, are not needed. Moreover, environmental lead levels in the United States provide no margin of safety to protect children; this is well-illustrated by the large number of children with lead levels in the toxic range. These effects of lead have been reviewed elsewhere in detail (ATSDR, 1988; EPA, 1986; EPA, 1989) and are only briefly summarized in this discussion.

High levels of lead in the body cause encephalopathy manifested by convulsions, mania, confusion, somnolence, or coma; if untreated, lead poisoning often results in death. Encephalopathy has been reported in persons with blood lead levels as low as 80 ug/dL (EPA, 1986). Since blood lead levels of 80 ug/dL can cause frank encephalopathy, it is not surprising that lower levels cause adverse effects on the central nervous system. In addition, lead affects the kidney, reproductive system, hematopoietic system, and virtually all other systems of the body.

Particularly disturbing are the following effects of lead exposure: 1) neurobehavioral effects of lead (including electrophysiologic changes) that occur at blood lead levels at least as low as 10 to 15 ug/dL; 2) reduced gestational age and reduced weight at birth that occur at levels at least as low as 10 to 15 ug/dL; 3) reduced growth rates up to 7 to 8 years of age that occur at levels at least as low as 10 to 15 ug/dL; 4) effects on heme metabolism starting at levels of about 15 to 20 ug/dL; and 5) effects on vitamin D

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metabolism starting at levels of about 15 to 20 ug/dL (ATSDR, 1988; EPA, 1986; EPA, 1989). Some studies have even indicated effects at levels below 10 ug/dL; some effects appear to have no threshold. Millions of children have blood lead levels above or near these values.

In addition, studies on health effects of lead exposure during the past 20 years have produced a consistent trend: the more that is learned about lead's effects on children and the fetus, the more concern is generated by lower and lower blood lead levels. The lowest observed adverse effect level (LOAEL) continues to drop. Blood lead levels formerly considered safe, or without adverse effect, have now been clearly associated with adverse effects.

Although other health effects are of significant concern, a dominant focus of recent studies of lead is the effect of lead on central nervous system cognitive function (e.g., intelligence). When the results are viewed collectively, a series of both prospective and cross-sectional studies provide persuasive evidence of ad's effects on children's cognitive function at blood lead levels as low as 10 ug/dL (ATSDR, 1988; EPA, 1986; EPA, 1989). Blood lead levels of 10 ug/dL and above at age 2 years have been shown to result in a reduction of the General Cognitive Index at age 57 months. Most of the children studied had blood lead level pelow 15 ug/dL (Bellinger, 1991). Although researchers have not yet fully defined the impact of blood lead levels <10 ug/dL on central nervous system function, it may be that even these levels are associated with adverse effects that will be more clear as our research instruments become better. If there is a threshold for lead's effects, it is near zero.

In a recent long term follow-up study (Needleman, 1990), for children exposed to moderate lead levels during preschool years, the odds of dropping out of high school were seven times higher and the odds of a significant reading disability were six times higher than for children exposed to lower lead levels. In addition, these children had lower class standing, increased absenteeism, and lower vocabulary and grammatical-reasoning scores, even after controlling for other covariates. The magnitude and persistence of these impacts on ability to learn and perform well in school suggest that lead exposure may have a significant deleterious effect on how well a child will function in society.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). The nature and extent of lead poisoning in children in the United States: a report to Congress, 1988. Atlanta: U.S. Department of Health and Human Services, 1988.



Bellinger D, Sloman J, Leviton A, Rabinowitz M, Needleman H, Waternaux C. Low-level exposure and children's cognitive function in the preschool years. Pediatrics 1991;57:219-227.

Bornschein RL, Succop PA, Krafft KM, et al. Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: Hemphill DD, ed. Trace substances in environmental health. Columbia, Mo: University of Missouri, 1986: 322-32.

Centers for Disease Control (CDC). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: U.S. Department of Health and Human Services, 1985; CDC report no. 99-2230.

Chisholm JJ, Jr. Interrelationships among lead in paint, housedust, and soil in childhood lead poisoning: The Baltimore experience. In, Davies BE, Wixson BG, eds. Lead in soil: issues and guidelines. Northwood, U.K.:Science Reviews Limited, 1988: 185-93.

Environmental Protection Agency (EPA). Air quality criteria for lead. Research Triangle Park, N.C.: Office of Health and Environmental Assessment, 1986; EPA report no. EPA/600/8-83/028aF.

Environmental Protection Agency. Lead in gasoline (Quarterly summary of lead phasedown reporting data), March 8, 1990.

Environmental Protection Agency (EPA). Supplement to the 1986 EPA air quality criteria for lead - volume 1 addendum. Research Triangle Park, N.C.: Office of Health and Environmental Assessment, 1989; EPA report no. EPA/600/8-89/049a.

Gilbert C, Tuthill RW, Calabrese EJ, et al. A comparison of lead hazards in the housing environment of lead poisoned children versus nonpoisoned controls. J Environ Sci Health 1979;A14(3), 145-68.

Levin R. Reducing lead in drinking water: a benefit analysis. Washington, DC: Office of Policy, Planning and Evaluation, 1986; EPA report no. EPA/230/09-86/019.

Needleman HL, Schell A, Bellinger D, et al. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. N Engl J Med 1990;322:83-8.

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APPENDIX II

BENEFITS OF PREVENTING LEAD EXPOSURE IN THE UNITED STATES AND COSTS AND BENEFITS OF LEAD-BASED PAINT ABATEMENT

Lead exposure among U.S. children has been estimated to cost society billions of dollars annually (e.g., Levin, 1986). For this Strategic Plan, we have developed a new benefits analysis, taking into account recent data on the effects of lead on children and fetuses. In addition, we have developed an example of a cost-benefit analysis for the abatement of lead-based paint in pre 1950 housing. We based this analysis on data from three studies conducted between 1983 and 1988; information on the costs and benefits of abatement will have to be continually updated as newer information becomes available. The Department of Housing and Urban Development and others are attempting to develop new and more effective abatement practices.

THE BENEFITS OF PREVENTING LEAD EXPOSURE AMONG CHILDREN AND FETUSES

This analysis will focus on the benefits of preventing exposure to lead among children and fetuses. The benefits of reducing lead exposure of persons already being exposed are likely to be substantial, but they are difficult to quantify. For example, we do not know how long a child needs to have an elevated blood lead level to develop cognitive deficits, although presumably longer durations of exposure have greater and possibly more longlasting effects. Therefore, the benefits of reducing exposure in already-exposed persons will not be included in the main portion of this analysis, although they will be included in the sensitivity analysis. For purposes of this analysis, the benefits of preventing exposure to lead in children and fetuses are the avoided costs that would have been incurred had exposure occurred. The benefits for which we provide monetary values are 1) reduction in medical care costs incurred by poisoned children, 2) reduction in special education costs for poisoned children, 3) reduction in future lost productivity due to cognitive deficits in children, and 4) reduction in neonatal mortality due to prenatal lead exposure.

The above benefits are only a few of the benefits of preventing lead exposure. Many benefits cannot be described in monetary terms, (e.g., avoiding the emotional costs to families of having a lead-poisoned child). Other benefits, such as preventing lead's effects on children's stature, hearing, vitamin D metabolism, and blood production, will not be explored in this analysis. The reason is not that they are unimportant, particularly when summed over millions of children; rather, it reflects the absence of methods for estimating appropriate monetary values for these effects. We also have not evaluated the potential contribution of lead to juvenile delinquency (Needleman, 1989), the administrative costs of personal injury lawsuits, the improvement in property values from



improved housing conditions resulting from abatement, or the effects of lead on adults, such as increased rates of hypertension, stroke, and cardiovascular disease. By not including these effects, we grossly underestimate the costs of lead exposure to society.

The benefits evaluated in this analysis fall into two categories. The first category consists of only the benefits that will be achieved for children whose blood lead levels are prevented from rising above a certain threshold; avoided medical and special education costs are estimated only for those children who would have had blood lead levels ≥25 ug/dL. These costs are presented as the average cost for each child in this category; the figure derived takes into account that not all children with blood lead levels ≥25 ug/dL will need chelation therapy or special education. The second category consists of benefits of preventing increased blood lead levels in children no matter what their initial levels are. For example, intellectual deficits result over a broad range of blood lead levels. We estimated the avoided costs due to the effects of lead on intellectual functioning for preventing increases of 1 ug/dL in blood lead level, regardless of the child's starting blood lead level. The benefits of reducing maternal blood lead levels (i.e., decreased infant mortality) are also included in this latter category.

The Benefits of Preventing Children from Developing Blood Lead L^vels ≥25 ug/dL

Medical costs: We assume, per the 1985 statement by the Centers for Disease Control, Preventing Lead Poisoning in Young Children, that children identified with blood lead levels ≥25 ug/dL will receive medical attention. Estimates of the medical care these children would need are based on data from Piomelli et al. (1984). We updated cost data from the regulatory impact analysis prepared by the Environmental Protection Agency (EPA) for reducing lead in gasoline (Schwartz et al., 1985) to 1989 using data from the medical care component of the Consumer Price Index.

Follow-up tests and administrative expenditures for all children whose blood lead levels are ≥25 ug/dL will total \$148 per child. Previous benefit analyses have used data from Piomelli et al. indicating that 70% of children with blood lead levels ≥25 ug/dL will have erythrocyte protoporphyrin levels ≥35 ug/dL and will receive provocative disodium calcium-edetate (EDTA) testing and follow-up. Provocative chelation requires a one-day hospitalization and one physician visit and is assumed to cost \$740. These same children will require a further series of follow-up tests and physician visits totaling \$444.

Five percent of children with blood lead levels ≥ 25 ug/dL will receive chelation therapy (Schwartz et al., 1985), requiring five days of hospitalization, several physician visits, laboratory testing and a neuropsychological evaluation. Half of these (2.5%) will require a second chelation therapy because their blood lead levels will rebound to ≥ 25 ug/dL. Half of these (1.25%) will require a third round of chelation therapy. (Therefore, an average of .0875 chelation therapies will be required for every child with a blood lead level ≥ 25 ug/dL.) The cost of each chelation therapy is estimated to be \$3,700.



To estimate the average medical cost per child with a blood lead level ≥ 25 ug/dL, the costs are multiplied by the associated probabilities by using the following equation:

$$AMC = PFU(\$FU) + PEDTA(\$EDTA) + PCHEL(\$CHEL)$$

= 1.0(\\$148) + 0.70(\\$740 + \\$444) + 0.0875(\\$3,700) = \\$1,300

where AMC = Average medical costs for children ≥ 25 ug/dL

PFU = Probability of follow-up testing for children ≥25 ug/dL

\$FU = Cost of follow-up testing

PEDTA = Probability of receiving provocative EDTA testing and

follow-up

\$EDTA = Cost of EDTA testing and follow-up

PCHEL = Probability of receiving chelation therapy

\$CHEL = Cost of chelation therapy

Therefore, the total medical cost that can be avoided by preventing a child from developing a blood lead level above 24 ug/dL is \$1,300.

Costs of special education: Children with high blood lead levels are more likely to have decreased school performance and require reading or speech therapy or psychological assistance. The costs of such treatment can be substantial. In a 3-year follow-up of children with high and low blood lead levels, de la Burde and Choate (1975) reported a relative risk of 7 for poor academic progress and a relative risk of 4 for repeating a grade. In addition, they reported that cognitive effects persisted for at least 3 years. Bellinger et al. (1984) reported that an excess of 17% of children with high blood lead levels were receiving daily assistance outside the classroom. Needleman et al. (1990) recently reported an odds ratio of 5.8 for reading disability among the children in their high lead group. Lyngbye et al. (1990) reported an odds ratio for learning disability of 4.3 for children with tooth lead levels above 16 parts per million (ppm).

On the basis of these reports and previous benefits analyses (Schwartz et al., 1985), we assume that 20% of children with blood lead levels ≥25 ug/dL will require special education (defined as assistance from a reading teacher, school psychologist, or other specialist) for an average of 3 years. Costs for part-time special education have been estimated by Kakalik et al. (1981) to be \$5,827 per year (updated to 1989 by using the Consumer Price Index). Because costs would be incurred over 3 years, costs in years 2



and 3 are discounted at $5\%^*$ to the year special education begins. The average special education costs for children with blood lead levels ≥ 25 ug/dL in year 1 are computed by using the following equation:

ASEC = (PSE)(\$SE)= (0.20)(\$5,827) = \$1,165

where ASEC = Average special education costs

PSE = Probability of requiring special education

\$SE = Cost of special education

Discounting years 2 and 3 by 5% results in total special education costs of \$3,331 per child with a blood lead level \geq 25 ug/dL.

The Benefits of Preventing a 1 ug/dL Increase in the Blood Lead Levels of Children

Most children with lead-related cognitive deficits do not require special education or other assistance; however, their losses can still be substantial in monetary terms. Impaired cognitive functioning and IQ decrements can reduce a person's productivity in society. In this benefits analysis, we use this loss in productivity as a proxy for the cost to society of cognitive impairment. This cost is clearly an underestimate because it puts no value on the losses sustained by the individual that are not reflected by decreased economic productivity. In addition, this analysis does not consider unearned income (e.g., interest, dividends), which would presumably be affected as the wage rate. We assume for this analysis that the benefits of reducing lead exposure on the cognitive functioning of children exhibit no threshold.

Figure 1 depicts the relationship between lead exposure and earnings. The first way lead exposure affects earnings is through its effect on IQ.

Lead has a direct effect on cognitive functioning, as measured by changes in IQ (pathway a). This reduction in IQ then has a direct effect on wage rate (pathway b), which affects



When costs or benefits occur in the future, they should be adjusted by discounting. The principle behind discounting is that there is a social as well as a personal preference for postponing costs and obtaining benefits as soon as possible. Therefore, dollars available in the future are less valuable than those available today. Mathematically, discounting future dollars can be thought of as the opposite of computing a return on an investment. Discounting, therefore, has the effect of reducing the numerical value of benefits or costs occurring in the future. For all calculations, we use a discount rate of 5% real (i.e., 5% above the rate of inflation).

lifetim arnings. Lead also affects earnings through its effect on educational attainment by redu IQ and by other effects, such as decreased attention span (pathway c). The effect of educational attainment on earnings is traceable through two main pathways. First, educational attainment is directly associated with wage rates and, therefore, with lifetime earnings (pathway d). Second, educational attainment is also associated with labor force participation (pathway e), which again has an effect on lifetime earnings.

The relationship between lead exposure and IQ (pathway a): Needleman and Gatsonis (1990) reported on a meta-analysis of the recent studies associating lead exposure with cognitive deficits. Although they reported only joint p values and partial r values, we used this information to perform a meta-analysis on effect size. We computed the estimated change in IQ for a 1 ug/dL change in blood lead for the six studies for which regression coefficients relating blood lead levels to IQ decrements were reported. Weighting by the inverse of the variance of each estimate, we estimate that each 1 ug/dL change in blood lead level results in a 0.25 point change in IQ.

The direct effect of IQ on wage rate (pathway b): A large body of literature exists on the relationship between IQ and wage rate. For example, in studies that examined the economic impact of increased schooling, it was important to control for differences in IQ; thus, the marginal impact of IQ on wage rate was estimated. In a review of the literature, estimates of the direct effect of IQ on wage rate (pathway b) ranged from a 0.2% to a 0.75% change in wage rate for each one IQ point change (Barth et al., 1984).

Structural equations modeling can be used to estimate the impact of multiple variables on an outcome of interest. Griliches (1977) used structural equations modeling and estimated the direct effect of IQ on wage rate to be slightly more than 0.5% per IQ point. Because this method has conceptual advantages and 0.5% is roughly the median estimate in the review by Barth et al. (1984), we used this value in these benefits estimates.

The impact of lead exposure on educational attainment (pathway c): From Needleman et al. (1990) and Needleman and Gatsonis (1990), it is possible to estimate the change in years of schooling attained per 1 IQ point change. The regression coefficients for the effect of tooth lead on achieved grade in those studies provide an estimate of current grade achieved, not of expected grade. Some of the children in those studies were, however, in college at the time of data collection and were expected to attain a higher grade. After adjusting the published results for the fact that a higher than reported percentage of the children with low tooth lead were likely to be attending college, we estimated a 0.59 year difference in expected maximum grade achieved between the high and the low exposure groups. We assumed that educational attainment scales with blood lead levels in proportion to IQ. The difference in IQ score between the high and the low exposure groups was 4.5 points. By dividing .59 by 4.5, we estimate that, the increase in blood lead level that reduces IQ by one point, reduces years of schooling achieved by 0.131 years.



Education and wage rate (pathway d): Studies that allow estimates of the relationship between educational attainment and wage rate (pathway d) are less common than those assessing the direct effects of IQ on wage rate. Chamberlain and Griliches (1977) estimated that a one year's increase in schooling would increase wages by 6.4%. In a model with similar specifications, Olneck (1977) reported a 4.8% increase. In a longitudinal study of 799 subjects for 8 years, Ashenfelter and Ham (1979) reported that an extra year of education increased the average wage rate by 8.8%. We have taken 6% as a reasonable and slightly conservative estimate of the effect of a year of schooling on wage rate.

Education, labor force participation, and earnings (pathway e): In addition to affecting wages, lead exposure is likely to affect participation in the labor force for several reasons. Labor force participation is correlated with failure to graduate from high school, principally through higher unemployment rates and earlier retirement ages. Lead exposure is also strongly correlated with attention span deficits and other effects which would also be likely to reduce labor force participation.

The differences in labor force participation between high school graduates and nongraduates were obtained from an analysis of the data in the 1978 Social Security Survey of Disability and Work by Cropper and Krupnick (1989), which controlled for age, marital status, number of children, race, region, and other socioeconomic and medical variables. We have estimated, using their regression coefficients, that average participation in the labor force is reduced by 10.5% for persons who fail to graduate from high school (pathway e). It is possible that this analysis overcontrols for other factors in estimating the effect of schooling. For example, high school drop-outs are more likely to have occupations with a higher risk of disability, which was also included as an independent variable in the regression analysis. Using the 1978 Current Population Survey, stratified by age groups between 25 and 65 years, we found that the mean number of hours worked in the previous year was 20% lower for persons with less than a high school education than the number worked by those with a high school education and those who graduated from high school. This difference results from reduced participation in the labor force and reduced hours worked by participants, and suggests that lead exposure sufficient to cause a 1 IQ point decrease would decrease expected earnings by about twice as much as reported by Cropper and Krupnick. To be conservative, we have used the results derived by using the regression models in Cropper and Krupnick as the estimate in this analysis. Using the study of Needleman et al. (1990), we estimate that lead exposure sufficient to cause a 1 point reduction in IQ would result in a 4.5% increase in the risk of failing to graduate (pathway c').

Lifetime earnings: Annual lifetime earnings benefits achieved by preventing a 1 ug/dL increase in a ch. I's blood lead level are computed as the net present value of the increased earnings expected from preventing the increase, discounted to age 6. To calculate the net present value of lifetime earnings, a number of assumptions are required. First, dollars available in the future must be discounted (see footpote, page 4).



The second assumption is that the real wage growth in the future will be 1% per annum from the 1987 distribution of inchaes. (Histor ally, real wage rates have increased approximately 2% per annum; however, in however, in assumes that growth rate has fallen.) This assumption is conservative because (1), it assumes that the same percentage of the work force will have a college education in the future as in 1987, and (2) it assumes the 1987 ratio of female to male earnings will remain unchanged, whereas the ratio has increased from 0.6 to 0.7 in the past 15 years and is expected to continue to increase in the future. On the other hand, this assumption is not conservative because it assumes that women will participate in the labor force at the same rate as in 1987, hereas their participation is likely to increase. If women continue to earn less than men, the real overall wage rate may not grow as quickly as 1% per year.

A third assumption made in calculating the net present value of lifetime earnings concerns labor force participation and the value placed on the productivity of nonparticipants. Many adults do not participate in the work force at all during their potential working years. The largest group are women who remain at home doing housework and child rearing. There is no consensus on how to put a monetary value on this nonmarket productivity. Work in the home has been valued in economic studies by using either the opportunity cost (the value of foregone income) or the market value of substitute labor for this work. The opportunity cost is usually taken as the average wage earned by persons of the same age, sex, and educational level. This may be too high, as the employed members of these cohorts tend to have more work experience, more training, and more relevant education than those who remain at home. The estimate based on the market value of substitute labor is often too low, as many of the substitute workers have less education than the persons they would replace. The most appropriate value is likely to be between these two estimates.

Given an estimate of the value of this nonmarket work, an additional assumption must be made about whether the impact of lower IQ on nonmarket productivity is the same as on market wages. There appears to be an association between maternal IQ and child's IQ, which is unlikely to be entirely hereditary. Moreover, in recent lead studies the Home Observation for Measurement of the Environment (H.O.M.E.) score (a measure of the quality of the home rearing environment) has been positively correlated with the mother's and the child's IQs (Bellinger et al., 1984). These findings suggest that IQ has an impact on nonmarket work, at least on the child-rearing component. For this analysis, we have taken the value of lost productivity due to lead exposure for nonparticipants in the labor force as half the value for employed workers.

Data for calculating the values of the expected lifetime earnings of an average child in the United States, under these assumptions, were obtained from 1987 earnings profiles from the U.S. Bureau of the Census. To compute the average lifetime earnings, we assumed that the numbers of men and women in the population would be equal, and part-time workers and non-labor force participants would earn half as much as average full-time workers. The net present value of average lifetime earnings per child,



discounted to age 3, is estimated to be \$260,000. The average child born into a housing unit 1 year after abatement is 4 years younger than the child currently occupying the unit; discounting that cl. ld's lifetime earnings to today's value yields a net present value of \$223,000.

Total earnings benefits: Figure 1 shows the pathways through which lead exposure affects total earnings benefits. The lower case letters a to e correspond to the pathways on Figure 1., on page 25.

We used the following equations to calculate the total lost wages attributable to reductions in earnings because of lead exposure:

1. The estimated change in wage rate for a 1 ug/dL change in blood lead level can be expressed as follows:

$$a*b = .25*.5\% = .125\%$$

where a = estimated change in IQ for each 1 ug/dL change in blood lead level (.25 IQ points per 1 ug/dL change in blood lead level)

b = estimated percentage change in wages for a 1 IQ point change (.5% wage change per IQ point)

2. The average change in wage rate from the decreased educational attainment resulting from lead exposure can be expressed as follows:

$$a*c*d = .25*.131*6\% = .197\%$$

where c = estimated change in grade attained for a 1 IQ point change (.131 years schooling per 1 IQ point change resulting from lead exposure)

d = estimated percentage change in wage rate for a 1 years change in grade attained (6% per year of schooling)

3. The average change in wage rate from decreased labor force participation from failure to graduate from high school can be expressed as follows:

$$a*c'*e = .25*4.5\%*10.5\% = .118\%$$

where c'= estimated change in the probability of graduating from high school for a 1 IQ point change resulting from lead exposure (4.5% increased probability of failure to graduate for each 1 point decrease in IQ)

- e = estimated percentage change in labor force participation because of failure to graduate from high school (10.5% decrease in labor force participation because of failure to graduate)
- 4. Therefore, the change in the expected present value of lifetime earnings from a 1 ug/dL change in blood lead levels can be expressed as follows:

```
\Delta E = E[(ab) + (cd) + (c'e)]
= $260,000[(.125% + .197% + .118%)]
= $260,000*.441% = $1,147*
```

where ΔE = the expected change in lifetime earnings from exposure to lead E = the net present value of lifetime earnings

We estimate, therefore, that prevention of an increase of 1 ug/dL in a child's blood lead level will produce a net present value benefit of \$1,147 per child. We again note that lost income is a clear underestimate of cognitive impairment, reduced educational attainment, and reduced labor force participation.

The Benefits of Preventing Prenatal Exposure to Lead

Prenatal lead exposure has been linked with reduced gestational age, lower birth weight, and decreased cognitive functioning, even in children exposed to low-to-moderate maternal blood lead levels (Dietrich et al., 1987). In this analysis, we assess only the impact on mortality of low gestational age due to lead exposure, since the data supporting the relationship between prenatal lead exposure and gestational age are stronger than the data supporting the relationship between lead exposure and low birth weight. In addition, to avoid the possibility of counting some infants in both prenatal and postnatal estimates, we did not assess the consequences of cognitive damage.

Prenatal exposure has also been linked to stillbirths in a number of studies (Vimpani et al., 1990), but we have not computed any benefits of avoiding fetal loss because the evidence is not complete and no study provides a dose-response function. We also have not computed costs from hospitalizations for premature and low birthweight infants.

These omissions result in an underestimate of the benefits of reducing prenatal exposure to lead.

The impact of prem tal exposure on mortality: We used data from the Linked Birth and Infant Death Record Project (National Center for Health Statistics) to estimate infant



The numbers shown are based on calculations using the most precise numbers possible. Because of rounding, there may be small differences between the numbers shown and those obtained by performing the calculations described.

mortality as a function of gestational age. The impact of prenatal lead exposure on gestational age is obtained from Dietrich et al. (1987). These estimates yield a predicted reduction of 10^4 (or 0.0001) in risk of infant mortality for each 1 ug/dL reduction in maternal blood lead level. In this analysis, we assume that the relationship between neonatal mortality and low gestational age is the same whether it results from prenatal lead exposure or from all other causes of low gestational age.

Valuing reductions in mortality: Placing a monetary value on reductions in mortality is highly controversial. The U.S. Department of Transportation has used lifetime wages (human capital approach) as a proxy, an approach common in litigation as well. This approach has obvious faults. For example, the value of reducing early mortality among retired persons or housewives is not zero, even though they may not be expected to earn wages. Because this approach underestimates the value of human life by approximating its value with the economic productivity of an individual, most economists prefer the willingness to pay method for valuing reductions in mortality.

Numerous methods for valuing people's willingness to pay for reducing their risk of mortality have been employed. The two most common methods include surveys that present realistic scenarios of trade-offs between expenditures and mortality risks or contingent valuation studies (Jones-Lee et al., 1985; Gegax et al., 1985) and assessments of market transactions that reveal implicit trade-offs between risk and dollars (e.g., Thaler and Rosen, 1976; Smith, 1976; Viscusi, 1978; Viscusi and O'Connor, 1984). Estimates resulting from these studies range from \$500,000 to \$9 million per statistical life, with most estimates falling between \$1 million and \$5 million (Violette and Chestnut, 1989). We have taken \$3 million per statistical life as the best estimate of the willingness to pay to avoid excess mortality risk.

Under these assumptions, the monetary benefit associated with reducing infant mortality is (0.0001) (\$3,000,000) or \$300 per ug/dL increase in blood lead level prevented for each pregnant woman.

Total Benefits of Preventing Lead Exposure

On the basis of the above analyses, the benefits of preventing a child's blood lead level from reaching 25 ug/dL are \$4,631 for avoided medical and special education costs. The increased productivity to be expected from preventing a l ug/dL increase in a child's blood lead level is \$1,147. Clearly, the greater the prevented increase in blood lead level, the greater the benefits; for the individual child, preventing the blood lead level from exceeding 24 ug/dL results in maximum benefits. The average benefits of preventing a l ug/dL increase in the blood lead level of a pregnant woman are \$300.

ABATEMENT OF HO

In this section we describe us costs and effectiveness of lead-based paint abatement.



Effectiveness of Lead-Based Paint Abatement in Reducing Lead Exposure

Studies have shown that abatement of lead-based paint in housing is effective in reducing children's blood lead levels (Kennedy, 1978), but quantitative data on these reductions are limited. We obtained both cost of abatement and effectiveness data for three evaluations of the efficacy of abatement—a study by Rosen et al. in New York City (in press), a study from St. Louis (G. Copley, unpublished data), and a study from Massachusetts (Y. Amitai et al., unpublished data). These data are not necessarily representative of abatements as they are currently performed. However, because of the lack of other data, we used them for our cost-benefit analysis.

In a study on the use of bone lead measurements in New York City children, Rosen et al. (in press) reported on children who did not receive chelation therapy but who did have their homes abated. At 24 weeks, the children's blood lead levels had declined from an initial mean level of 29 ug/dL to 21 ug/dL. Abatement methods used in this study included scraping, spackling and repainting surfaces with deteriorating lead paint.

An unpublished study from the City of St. Louis Division of Health (G. Copley, unpublished data) reported that children who did not receive chelation and whose homes were abated experienced a mean reduction of 9.3 ug/dL in blood lead levels (from 43.9 to 34.2 ug/dL) measured 6 to 12 months after the abatement. Abatement consisted of scraping or encapsulating deteriorated surfaces.

An evaluation of data collected in 1984 and 1985 by the Massachusetts Childhood Lead Poisoning Prevention Program (Y. Amitai et al., unpublished data) examined the intraabatement and postabatement blood lead levels of children who received no chelation therapy. The purpose of the study was to examine the impact of abatement method on intraabatement blood lead levels when children were not relocated during abatement. Several abatement methods were employed, including dry scraping, sanding, and encapsulation. Mean blood lead levels 8 months postabatement decreased by 10.2 ug/dL (from 35.7 ug/dL to 25.5 ug/dL).

In these three studies, the approximate mean decrease in blood lead levels after abatement was 9 ug/dL for children in lead-contaminated housing and with initial blood lead level ≥ 25 ug/dL.

These studies only included children with blood lead levels ≥25 ug/dL. No data are available on the effects of abating homes of children with lower blood lead levels. In the studies, the mean decrease in a child's blood lead level with abatement was 25%. For the cost-benefit analysis presented here, we assume that the reduction in blood lead levels from abatement of homes of children with initial levels <25 ug/dL will be proportional to the reduction for children with lower blood lead levels. Thus, children with blood lead levels <25 ug/dL will experience a 25% decrease in blood lead levels



from lead-based paint abatement. Using estimates from models developed by EPA and others, we estimate that the mean blood lead level for children whose preabatement levels are between 10 and 24 ug/dL is 15 ug/dL (J. Schwartz, personal communication). For children whose blood lead levels are between 10 and 24 ug/dL, the mean decrease in blood lead levels expected from abatement is 3.75 ug/dL.

Costs of Lead-Based Paint Abatement

We contacted individuals associated with the abatement programs in New York City, St. Louis, and Massachusetts to ascertain the nature and approximate costs of the abatement methods used at the time that data for these studies were compiled. All three programs relied extensively on scraping, spackling, and repainting areas with deteriorated lead-containing paint. Encapsulation was less frequently used (only in Boston and St. Louis), and there was no replacement of doors, windows, or woodwork. Only deteriorated or damaged lead-containing surfaces were abated routinely in New York City and St. Louis, while Boston abatements included stripping of all chewable, accessible surfaces below 5 feet (e.g., window sills, baseboards, door frames), regardless of condition, if they contained lead. Costs for abating an average unit of 5 to 6 rooms for each of the cities were as follows: St. Louis - \$2,000, New York City - \$2,500, and Boston - \$1800 (inflated when necessary to 1989 prices by using the Consumer Price Index). These prices include abatement of common areas and exteriors when necessary and costs of materials, labor, insurance, overhead, whatever werker protection was employed, preparation of the unit before abatement, and cleanup. An average cost of \$2,100 will be assumed for these studies.

It should be noted that some currently recommended abatement methods and procedures are much more expensive than those discussed above. A cost-benefit analysis was not conducted for these more rigorous abatements because data on associated changes in blood lead levels are not available.

The investigators in New York City, St. Louis, and Massachusetts were questioned about the longevity of the effectiveness of these abatement methods--that is, about how long a relatively "lead-free" environment would be maintained in the home. In all three cases, the investigators reported that repoisoning after abatement was very infrequent (considerably less than 1% within a year). This does not address the problem of long-term effectiveness of the abatement, for example, 5 to 15 years after the original abatement is completed.

The average charge for home inspections in several lead poisoning prevention programs is \$97 per unit (K. O'Connor, C. Torres, H. Billingsly, personal communications), which we round to \$100. If we assume that 80% of pre-1950's housing contains leaded paint (Shier and Hall, 1977), the cost for an investigation per positive home is \$125. Therefore, the total cost of abatement is \$2,225 per unit. Several costs are not included in these estimates because they are more difficult to quantify or are extremely variable.



One source of costs is court proceedings, for example, when notices to landlords are challenged; another is for the dislocation of families from their homes and the effects on neighborhoods when landlords refuse to abate marginally viable housing. An additional cost results if 'amilies are relocated to alternative housing at program expense.

COST-BENEFIT ANALYSIS

In the following section, we present a cost-benefit analysis for abatement of an average house with lead-based paint built before 1950. In the analysis, we will use the data presented earlier on the costs and effectiveness of abatements performed in St. Louis, New York City, and Boston.

The benefits used in this analysis are likely to be substantial underestimates of the true benefits of abatement. In addition to the reasons for underestimation already discussed, a very important component of underestimation in this analysis is that we will not assign monetary value to the benefits of abating homes of children and pregnant women who already are currently being exposed to lead. This assumption may be unjustified for several reasons. First, children remaining in a lead-contaminated environment may need repeated courses of medical treatment for continued elevations in blood lead levels. Second, the blood lead levels of some of these children will increase further as a result of living in lead-contaminated homes, thereby increasing the probability that they will need medical care and special education and further reducing their future earnings. Third, decreasing the amount of time children and pregnant women have elevated blood lead levels will probably decrease the adverse effects from lead. Data are not ailable, however, to allow these benefits to be quantified. Therefore, this cost-benefit analysis is conducted under the assumption that we target homes for abatement in a high-risk area based on the home's containing lead-based paint and having been built before 1950. All benefits are accrued by children who will enter a high-risk age group in the house in the future and by fetuses potentially exposed to lead in the future. No attempt is made to target abatement to the homes of currently lead-poisoned children.

For this cost-benefit analysis, we use the following assumptions:

Assumptions:

Assumption 1: In general, children's exposure to lead-based paint and paint-contaminated dust and soil begins to increase when they become mobile and decreases as they practice less mouthing behavior. We will assume that children less than 10 months and greater than 6 years of age are unlikely to be poisoned by lead-based paint, regardless of their housing. Therefore, quantitative benefits can be assessed for children who are less than 10 months of age and are now living in the unit, for children who are likely to be born into or move into the abated unit, and women who will become pregnant while living in the abated unit.



Assumption 2: On average, there are 0.287 children per house built before 1950 (Pope, 1986). The average number of children less than 10 months old per pre-1950 housing unit is $(0.287 \text{ children } \times (9/72 \text{ months})) = 0.036 \text{ children}$.

Assumption 3: This analysis is performed for the average home built before 1950 which is painted with leaded paint.

Assumption 4: The average overall loss rate of housing, both rental and owner-occupied, built before 1950 is 1% per year (D. McGough, Department of Housing and Urban Development, personal communication). On the basis of this assumption, the median remaining life of existing housing stock built before 1950 is 68 years.

Assumption 5: A targeting strategy is employed that abates homes built before 1950 that contain lead-based paint, whether or not these homes currently contain children.

Assumption 6: Were the targeted homes not abated, some of the children who occupied them would have become lead poisoned. For these children the increase in blood lead level prevented by abatement is 9 ug/dL. For children who would not have become poisoned, the average prevented increase in blood lead level is 3.75 ug/dL.

has sumption 7: On the basis of data from Cincinnati, the difference in blood lead levels nong pregnant women in lead-contaminated 19th century housing and those in lead-free public housing is 2.13 ug/dL (R. Bornschein, personal communication). On the basis of these data, we assume that abating a unit results in the prevention of a 2.13 ug/dL increase in the blood lead levels of pregnant women.

Assumption 8: Almost 6 million children under 7 years of age live in pre-1950 housing with high levels of lead in paint (ATSDR, 1988). Of these, 0.2 million, or 3.4%, have blood lead levels above 25 ug/dL. Thus, for this analysis we will assume that abatement will prevent blood lead levels ≥25 ug/dL in the 3.4% of children who would be expected to develop them otherwise and will prevent levels between 10 and 24 ug/dL in the rest. Thus, the average prevented increase in blood lead level for a child living in a house contaminated with lead-based paint is:

$$(0.034)$$
 (9 ug/dL) + (0.966) (3.75 ug/dL) = 3.93 ug/dL

Assumption 9: An average of 0.045 infants below age 1 year are present in housing units built before 1950 (Pope, 1986). We will therefore assume that 0.045 children are born into each unit each year after abatement and that 0.045 represents the proportion of pregnant women in such houses each year. Abatement prevents an increase of 2.13 ug/dL in the blood lead level of a pregnant woman. Thus, the average prevented increase in blood lead levels in pregnant women in abated housing is:

$$(0.045)$$
 $(2.13) = .096$ ug/dL



Assumption 10: The medical costs avoided by preventing a child born into a unit the year after abatement from developing a blood lead level ≥25 ug/dL is \$1,069 (discounted to 4 years into the future, since we assume costs are incurred at age 3 years). The avoided special education costs are \$2,365 (discounted to 7 years into the future). For each 1 ug/dL blood lead level increase prevented in a child, \$1,085 in lost earnings is avoided.

Assumption 11: Each avoided increase of 1 ug/dL blood lead in a pregnant woman by abating the unit the year before she becomes pregnant, when discounted to 1 year in the future, results in an average savings of \$286 from the prevention of infant mortality.

Assumption 12: A discount rate of 5% is used.

Assumption 13: Analysis is done for a set time. Both benefits and costs are discounted to that year.

Assumption 14: Benefits can be assessed for each cohort of children entering the home. Because we assume that the average remaining lifespan of housing units built before 1950 is approximately 68 years, benefits are calculated for 68 cohorts of children, with benefits being discounted appropriately.

Assumption 15: The discounted total value of benefits for children is equal to the sum of the benefits accruing for current resident children less than 10 months of age and the benefits accruing to children who will move into or be born into the residence in the future. These are the benefits of avoiding medical and special education costs and increasing earnings.

Assumption 16: Benefits are also accrued by reducing blood lead levels in pregnant women who will live in the house in the future.

The following equations summarize this information. In these calculations, figures are only presented up to 4 decimal places. As a result, an attempt to duplicate the calculations performed will result in rounding errors; final values are based on the most precise figures possible.

1. The proportion of children now living in the home who will accrue benefits from the avoidance of medical and special education costs can be expressed as follows:

$$f = g*h$$

= .034*.036 = .0012

where f = average number of children per house less than 10 months of age living in pre-1950 housing whose blood lead levels would be expected to rise above 24 ug/dL



- g = average proportion of children with blood lead levels above 24 ug/dL (.034; see Assumption 8, page 14)
- h = average number of children less than 10 months of age per housing unit built before 1950 (0.036; see Assumption 2, page 14)
- 2. The proportion of future children who will accrue benefits from avoidance of medical and special education costs can be expressed as follows:

$$i = g*j$$

= .034*.045 = .0015

where

- i = average number of children per house who will be born into the average pre-1950 house each year of the house's remaining lifespan and whose blood lead levels would be expected to rise above 24 ug/dL without abatement
- j = average number of pregnant women per house per year (.045; see Assumption 9, page 14)
- 3. The net present value of medical costs avoided through abatement is the sum of the avoided costs for children currently living in the unit plus the avoided costs for 67 cohorts of future children.

MED =
$$f*AMC + i*[AMC_1 + AMC_2 + ... + AMC_{67}]$$

= $.0012*\$1,300 + .0015*\$21,605.23$
= $\$1.59 + \$33.06 = \$34.65$

where

AMC = average medical costs for children with blood lead levels above 24 ug/dL the present year (\$1,300; see page 3)

AMC_n = average medical costs for children with blood lead levels above 24 ug/dL in year_n, discounted to the present year

4. The net present value of special education costs avoided through abatement is the sum of the avoided costs for children currently living in the unit plus the avoided costs for 67 cohorts of future children.

SEC =
$$f*ASEC + i*[ASEC_1 + ASEC_2 + ... + ASEC_{67}]$$

= $.0012*$3,331 + .0015*$47,783.47$
= $$...06 + $73.11 = 77.17

where ASEC = average special education costs for children with blood lead levels above 24 ug/dL in the present year (\$3,331; see page 4)



ASEC_n = average special education costs for children with blood lead levels above 24 ug/dL in year_n, discounted to the present year

5. The average net present value of lost earnings prevented by abatement for children less than 10 months of age currently living in the home is:

$$INC_c = h*k* \Delta E$$

= .036*3.93*\$1,147 = \$161.65

where INC_c = lost earnings prevented for current children less than 10 months of age living in lead-painted homes

k = average decline in blood lead levels of children from abatement (3.93 ug/dL; see Assumption 8, page 14)

 ΔE = change in earnings that can be attributed to a 1 ug/dL change in blood lead level (\$1,147; see page 9)

6. The net present value of lost earnings prevented through abatement for future children is the avoided costs for 67 cohorts of future children.

$$INC_f = j*k*[E_1 + E_2 + ... + E_{67}]$$

= .045*3.93*\$19,781 = \$3,497

where $INC_f =$ net present value of lost earnings prevented for future children who would live in pre-1950 homes

E_n = average present value decrease in earnings for each cohort, discounted to the present year

7. The net present value of lives saved from avoided mortality from reducing prenatal lead exposure can be expressed as follows:

LIFE =
$$i*[M_1 + M_2 + ... + M_{67}]$$

= .096*\$5,571.72 = \$553.22

where i = average prevented increase in a pregnant woman's blood lead level from abatement (.096 ug/dL; see Assumption 9, page 14)

M_n = average benefits from reduced fetal mortality of preventing a 1 ug/dL increase in blood lead level in year n, discounted to the present year

8. The total benefits of abatement are:

MED + SEC + INC_c + INC_f + LIFE
=
$$\$34.65 + \$77.17 + \$161.65 + \$3,497.00 + \$553.22 = \$4,323.70$$

Costs Versus Benefits of Abatement

We estimate that abating an average pre-1950 lead-painted home using the methods employed for the three studies described above earlier costs \$2,225, and the benefits over the lifetime of the home are \$4,323. Thus, abatement of a home results in a net benefit of \$2,098. This net benefit does not take into account any benefits sustained by a child who is already poisoned in the unit or the numerous benefits to which we could not assign monetary values.

This cost-benefit analysis provides an economic justification for a national program of abating lead-contaminated housing to prevent childhood lead poisoning. This analysis is conservative because a number of important benefits remain unquantified. Moreover, prevention of lead poisoning would be an important public health activity, even if no economic benefits could be demonstrated.

This analysis indicates what is needed for a rational national abatement program. Obviously, the better a plan for setting priorities for abatement can be targeted to homes likely to house children in the future, the greater the net benefits. Furthermore, if strategies can be developed for determining which homes are most likely to poison children, the efficiency and benefits of any abatement program will be markedly increased.

Sensitivity Analysis

Changing certain values may have an impact on the conclusions that are drawn from an analysis; consequently, we perform sensitivity analysis to test the impact of changing our assumptions. In this section, we report the results of sensitivity analyses aimed at testing whether the values of key variables significantly alter the conclusions that can be drawn from the study. Table 1 displays the results of the base case analysis along with the sensitivity analyses. Benefits are expressed in terms of net benefits-that is, the difference between the total benefits and the cost of abatement.

Changing the number of children per home: In the base case analysis, we assumed that abatement would not be targeted to homes with children; therefore, the average number of children per pre-1950 home was used in estimating the benefits of abatement. In this variation, we assume that abatement is conducted in communities with more children than the average. For this analysis, we assume that the average unit to be abated houses three times more children than the national average. Thus, we assume that, on the average, 0.108 (or 0.287*3*[9/72]) children less than 10 months of age now occupy the



unit and, for future cohorts, we assume that an average of 0.135 (or 0.045*3) children will be born into each unit each year. Under these assumptions, the net benefits are \$10,747 per unit. Alternatively, if we assume that five times the average number of children occupy these units than is the average for the nation, the net benefits are \$19,395 per unit.

Changing the discount rate: In the base case analysis we assumed a discount rate of 5%. If we discount all future benefits and costs by 3% the net benefits are approximately \$6,357. When all future benefits and costs are discounted by 7%, net benefits become \$404.

Changing the lifespan of houses: Assuming a 50-year lifespan rather than the median of 68 years reduces net benefits to \$1,866, and decreasing the lifespan of houses to 30 years reduces net benefits to \$1,212 per abated unit.

Changing the effectiveness of the abatement (ug.dL reduction): In the base case analysis, we assumed that children with blood lead levels ≥25 ug/dL experience a 9 ug/dL blood lead decline after abatement and that children with lower levels will have a 3.75 ug/dL decline. In this variation, we will assume that abatement is more effective than in the three studies from which we obtained data. We will assume that children with blood lead levels ≥25 ug/dL experience a 21.6 ug/dL decline (60% of the average baseline blood lead level) and that children with lower blood lead levels experience a 9 ug/dL decline. We also assume a proportionately greater decrease in the blood lead levels of pregnant women (5.11 ug/dL). Under this scenario, net benefits increase to \$7,992. This analysis implies that a more effective abatement method that results in an approximate average decline in the blood lead level of 9.4 ug/dL could cost as much as \$10,000 per unit and we could still expect to see net benefits from abatement.

Changing assumptions about the impact of abatement on children 9 months of age or older currently in lead-painted homes: In the base case analysis we estimated benefits only for those children currently living in the home who were less than 10 months of age. No benefits were assumed in the analyses for children above that age. In this variation, we will assume that all children 6 years of age and under living in an abated home will experience full benefits. In this case, the net benefits are \$3,290. If we assume that children between 10 months and 6 years of age receive only half the benefits of children less than 10 months, the net benefits are \$2,693.

These analyses show that targeting abatement to homes with children and improving the efficacy of abatement will result in greater net benefits. Furthermore, if strategies can be developed for determining which homes are most likely to poison children, the efficiency and benefits of any abatement program will be markedly increased.

ERIC

THE BENEFITS OF A NATIONAL EFFORT TO ABATE ALL PRE-1950 HOUSING UNITS WITH LEAD-BASED PAINT

In this analysis, we estimate the benefits from abating all homes in the United States.

- 1. Results of a study by Shier and Hall (1977) show that 80% of pre-1950 housing centains lead-based paint. Since there are 28,971,000 occupied pre-1950 housing units in the United States (U.S. Bureau of the Census, 1989), we estimate that there are 23,176,800 occupied pre-1950 housing units containing lead.
- 2. For this analysis, we use a cost estimate of \$2,225 per abatement. Therefore, the cost of abating all 23,176,800 units today would be \$51,568,380,000. Were the abatement conducted over the next 20 years (performing an equal number of abatements each year), the total present cost of abatement would be \$33,739,550,000.
- 3. We have estimated that the total benefits of abatement are \$4,323 per housing unit. If all abatements were performed now, the total benefits would be \$100,193,306,000. If abatements were conducted over the next 20 years, the total present value of the benefits would be \$61,742,270,000. (This number takes into account the fact that a house abated in the future has a shorter lifespan as a lead-free dwelling than a unit abated today; therefore, fewer cohorts of children would benefit.)
- 4. We have estimated that the net benefits of abatement (total benefits of abatement costs of abatement) are \$2,098. If all pre-1950 lead-painted housing units in the United States were abated today, the net benefits of abatement would be \$48,624,926,000. If units were abated over the next 20 years, the present value of the net benefits would be \$28,002,830,000.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). The nature and extent of lead poisoning in children in the United States: a report to Congress. Atlanta: U.S. Department of Health and Human Services, 1988.

Ashenfelter O, Ham J. Education, employment and earnings. Journal of Political Economy 1979;87:S99-S131.

Barth MC, Janney AM, Arnold F, Sheiner L. A survey of the literature regarding the relationship between measures of IQ and income. Washington, D.C.: ICF Inc., 1984.



Bellinger DC, Needleman HL, Leviton A, et al. Early sensory-motor development and prenatal exposure to lead. Neurobehavioral Toxicol Teratol 1984;6:387-402.

Centers for Disease Control (CDC). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: U.S. Department of Health and Human Services, 1985; CDC report no. 99-2230.

Chamberlain G, Griliches Z. More on brothers. In: Taubman P, ed. Kinometrics: determinants of socioeconomic success within and between families. Amsterdam: North Holland Publishing, 1977:97-124.

Cropper M, Krupnick AJ. The social costs of chronic heart and lung disease. Discussion paper QE 89-16. Quality of the Environment Division, Resources for the Future, August, 1989.

de la Burde B, Choate MS Jr. Early asymptomatic lead exposure and development at school age. J Pediatr 1975;87:637-42.

Dietrich KN, Krafft KM, Bornshein RL, et al. Low level fetal lead exposure effect on neurobehavioral development in early infancy. Pediatrics 1987;80:721-30.

Deitrich KN, Krafft KM, Shukla R, Bornschein RL, Succop P. The neurobehavioral effects of early lead exposure. In: Schroeder SR, ed. Toxic substances and mental retardation: neurobehavioral toxicology. Washington, D.C.: American Association of Mental Deficiency, 1987:71-95 (Monograph No. 8).

Gegax D, Gerking A, Schulze W. Perceived risk and the marginal value of safety. Report to the U.S. Environmental Protection Agency. 1985.

Griliches Z. Estimating the returns to schooling: some econometric problems. Econometrica 1977;45:1-22.

Jones-Lee MW, Hammerton M, Phillips PR. The value of safety: results of a national sample survey. Economic Journal 1985;95:49-72.

Kakalik JS. The cost of special education. Rand Corporation Rep. 1981, N-1791-ED.

Kennedy FD. The childhood lead poisoning prevention program: an evaluation. A report for the Centers for Disease Control. 1978.

Levin R. Reducing lead in drinking water: a benefit analysis. Washington, DC: Environmental Protection Agency (EPA), 1986; EPA report no. 230-09-86-019.



Lyngbye T, Hanson O, Trillingsgaarb A, Beese I, Grandejean P. Learning disabilities in children: significance of low-level lead-exposure and confounding factors. Acta Paediatr Scand 1990;79:352-60.

Needleman, HL. The persistent threat of lead: a singular opportunity. Am J Public Health 1989;79:643-45.

Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. JAMA 1990;263:673-78.

Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. N Engl J Med 1990;322:83-8.

Olneck M. On the use of sibling data to estimate the effects of family background, cognitive skills, and schooling: results from the Kalamazoo Brothers Study. In: Taubman P, ed. Kinometrics: determinants of socioeconomic success within and between families. Amsterdam: North Holland Publishing Company 1977:125-62.

Piomelli S, Rosen J, Chisolm JJ, Graef J. Management of childhood lead poisoning. J Pediatr 1984;4:523-32.

Pope A. Exposure of children to lead-based paints. Research Triangle Park, N.C.: U.S. Environmental Protection Agency (EPA), 1986; EPA report no. 68-02-4309.

Rosen JF, Markowitz ME, Bijur PE, et al. Sequential measurements of bone lead content by L-X-ray fluorescence in CaNa₂-EDTA treated lead-toxic children. Environmental Health Perspect (in press).

Schwartz J, Pitcher H, Levin R, Ostro B, Nichols AL. Costs and benefits of reducing lead in gasoline: final regulatory impact analysis. Washington, D.C.: U.S. Environmental Protection Agency (EPA), 1985; EPA report no. 230-05-85-006.

Shier DR, Hall WG. Analysis of housing data collected in a lead-based paint survey in Pittsburgh, Pennsylvania, Part 1. Washington, D.C.: National Bureau of Standards, 1977.

Smith RS. The Occupational Safety and Health Act. American Enterprise Institution for Public Policy Research, 1976.

Thaler R, Rosen S. The value of saving a life: evidence from the labor market. In: Taleckji NE, ed. House production and consumption. New York: Columbia University Press, 1976.

ERIC

U.S. Bureau of the Census. Statistical abstracts of the United States: 1989. 109th ed. Washington, D.C., 1989.

Vimpani G, Baghurst P, McMichael AJ, Robertson E, Wigg N, Roberts R. The effects of cumulative lead exposure on pregnancy outcome and child development during the first four years. Presented at advances in lead research: implications for environmental health conference. Research Triangle Park, NC: National Institute of Environmental Health Sciences, 1990.

Violette DM, Chestnut L. Valuing risks: new information on the willingness to pay for changes in fetal risks. Washington, D.C.: U.S. Environmental Protection Agency (EPA), 1989; EPA report no. 230-06-86-016.

Viscusi WK. Labor market valuations of life and limb: empirical evidence and policy implications. Public Policy 1978;26:359-86.

Viscusi WK, O'Connor CJ. Adaptive response to chemical labeling: are workers Bayesian decision makers? American Economic Rev 1984;74:942-56.



Table 1. Results of the base case cost-benefit analysis and sensitivity analysis, expressed as net benefits for abatement of the average pre-1950 home with lead-based paint, discounted to the present. (Net benefits = total benefits - cost of abatement.)

Description of Analysis	Net Benefits
Base case analysis (see text for assumptions)	\$ 2,098
Sensitivity analyses	
Number of children per home	
If increased 3-fold	10,747
If increased 5-fold	19,395
Discount rate of 5%	
If decreased to 3%	6,357
If increased to 7%	404
Life span of houses is 68 years	
If decreased to 50 years	1,866
If decreased to 30 years	1,212
Effectiveness of the abatement	
With 60% decrease in blood lead levels	7,992
Benefits accrue to children 10-72 months	
With 100% benefits	3,290
With 50% benefits	2,693



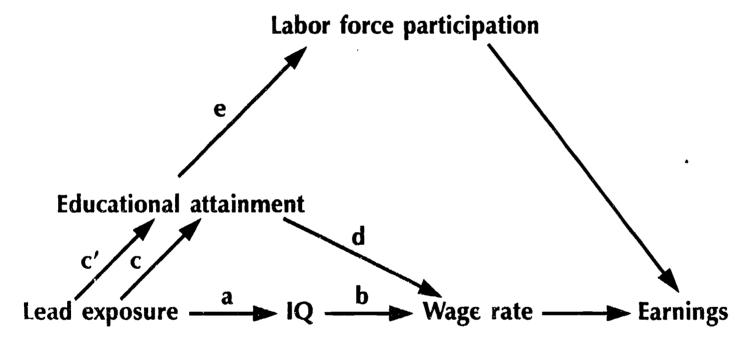


Figure 1. Effect of lead exposure on earnings.

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APPENDIX III

HISTORY OF CHILDHOOD LEAD POISONING PREVENTION PROGRAMS

The first cases of childhood lead poisoning from lead paint in housing were reported in 1892 in Australia. Although many severe cases of the disease were reported in subsequent decades in the United States, little effort was made to find additional cases until the 1950s, when caseworkers in a few large cities attempted to find lead-poisoned children. In 1966, Chicago began the first mass screening program, followed shortly by New York and other cities (Lin-Fu, 1980).

The Lead-Based Paint Poisoning Prevention Act, passed in 1971, initiated a national effort to identify children with lead poisoning and abate the sources of lead in their environments. For most years of this program, Federal funds appropriated under this Act were administered by the Centers for Disease Control (CDC). More than \$89 million were distributed, and over a quarter of a million children were identified with lead poisoning and received referrals for environmental and medical intervention.

In 1981 the Omnibus Budget Reconciliation Act amended Title V of the Social Security Act, which had authorized the Maternal and Child Health (MCH) Services Program since 1935. The amendment created the MCH Services Block Grant Program and consolidated many categorical programs, including that for childhood lead poisoning prevention, into the Block Grant. In 1982, the administrative responsibility for the Lead-Based Paint Poisoning Prevention Act was transferred to the Office of Maternal and Child Health (now the Maternal and Child Health Bureau) of the Health Resources and Services Administration.

Under the provisions of the MCH Services Block Grant Act, each State decides how to use these Federal funds. Data on whether these funds are used to support childhood lead poisoning prevention activities has not been reported to the Federal government. The 1989 Omnibus Reconciliation Act includes a requirement for State MCH Block Grant Programs to be consistent with the Public Health Service Year 2000 Objectives for the Nation and to submit an annual report with specified content in a standardized format. Since reduction of the numbers of children with lead poisoning is likely to be included as a Year 2000 Objective, more information on childhood lead poisoning prevention activities funded by the MCH block grant is anticipated.

The Lead Contamination Control Act of 1988 authorized \$20 million for Fiscal Year 1989, \$22 million for Fiscal Year 1990, and \$24 million for Fiscal Year 1991 for CDC to administer a childhood lead poisoning prevention grant program. Under this law, \$4



million were appropriated in Fiscal Year 1990, and \$8 million were appropriated in 1991. The President's budget for 1992 includes \$14.95 million for this program. The majority of this money will be provided as grants for State and local agencies to perform childhood lead screening, referral for medical and environmental follow-up, and education about lead poisoning in those communities with children with the highest blood lead levels. This money is directed at communities with large numbers of children with higher blood lead levels (e.g., $\geq 25 \text{ ug/dL}$). Although clearly many more States and communities need comprehensive programs to address childhood lead poisoning, CDC's current grant program is an important step in our effort to eliminate childhood lead poisoning.

The President's budget for FY 1992 also includes \$25 million for the HOME program, which will be administered by the Department of Housing and Urban Development (HUD). This program will assist low- and moderate-income private residential property owners to abate lead-based paint, and will be directed to homeowners with young children in high-risk housing. This program could provide a knowledge base for evaluating the effects of abatement.

REFERENCES

Lin-Fu J. Lead and children: a historical review. In: Needleman HL, ed. Low level lead exposure: the clinical implications of current research. New York: Raven Press, 1980:3-16.



APPENDIX IV

ORGANIZATIONS AND AGENCIES THAT COULD HELP PROMOTE AWARENESS OF CHILDHOOD LEAD POISONING

Table 1. Professional Organizations Thai Could Increase Practitioner Awareness of Childhood Lead Poisoning

Primary Care Physicians (family practice, internal medicine, pediatrics, and emergency medicine)

Ambulatory Pediatric Association

American Academy of Pediatrics

American Association of Family Physicians

American Board of Pediatrics

American College of Emergency Physicians

American College of Physicians

American Medical Association

American Medical Student Association

American Osteopathic Association

American Pediatric Society

American Society of Internal Medicine

Association of American Physicians

Association of American Indian Physicians

Association of General Practitioners/Family Physicians

Association of Medical School Pediatric Department Chairmen

Association of Program Directors in Internal Medicine

Federal Physicians Association

National Association of Residents and Interns

National Medical Association

North American Primary Care Research Group

Society of Teachers of Family Medicine

Society of General Internal Medicine

Public Health Physicians

American Association of Public Health Physicians

American College of Preventive Medicine

American Osteopathic College of Freventive Medicine

Association of Teachers of Preventive Medicine

Association of Preventive Medicine Residents



Table 1 (continued). Professional Organizations that Could Increase Practitioner Awareness of Childhood Lead Poisoning

Other Physician Speciality Organizations

American Association of Obstetrics and Gynecology American College of Occupational Medicine

Nurses

American Society of Pediatric Hematology/Oncology

American Nursing Association

American Academy of Nursing

American Association of Neuroscience Nursing

American Association of Occupational Nursing

American College of Nurse-Midwives

American Licensed Practical Nurses Association

American Nurses' Association

American Organization of Nursing Executives

Assembly of Hospital Schools of Nursing

Association of State and Territorial Directors of Nursing

Frontier Nursing Service

National Association of Hispanic Nurses

National Association of Pediatric Nurse Associates and Practitioners

National Association of Physician Nurses

National Association of Registered Nurses (State Associations)

National Association of School Nurses

National Association of Black Nurses

National Board of Pediatric Nurse Practitioners and Associates

Nurses Association of the American College of Obstetricians and

Gynecologists

Physician Assistants

American Academy of Physician Assistants Association of Physician Assistant Programs

Pharmacists

American Pharmaceutical Association
American Society of Hospital Pharmacists
National Association of Retail Druggists
National Pharmaceutical Association
National Pharmaceutical Foundation
State Boards of Pharmacy
State Pharmaceutical Associations



Table 1 (continued). Professional Organizations that Could Increase Practitioner Awareness of Childhood Lead Poisoring

Public Health Professionals

American College of Epidemiology

American Industrial Hygiene Association

American Public Health Association

Association of Schools of Public Health

Association of State and Territorial Directors of Public Health Education

Association of State and Territorial Health Officers

Association of State and Territorial Public Health Laboratory Directors

Association of University Programs in Occupational Health and Safety

Conference of Public Health Laboratorians

Conference of State Health and Environmental Managers

Council of State and Territorial Epidemiologists

Council on Education for Public Health

National Association of County Health Officials

National Coalition of Hispanic Health and Human Services Organizations

National Conference of Local Environmental Health Administrators

National Environmental Health Association

National Foundation of Rural Medical Care

National Rural Health Association

Society for Occupational and Environmental Health

United States Conference of Local Health Officials

World Federation of Public Health Associations

Other Health Organizations

American Indian Science and Engineering Society
American Industrial Health Council
Asian American Health Forum
Association of American Medical Colleges
Association of Minority Health Professions Schools
National Association of Community Health Centers
Society for Pediatric Research





Table 2. Other Organizations that Would be Interested in Educating the Public About Childhood Lead Poisoning

Maternal and Child Health

American Association of University Affiliated Programs for Persons with Developmental Disabilities

Association of Maternal and Child Health Programs

Be Healthy, Inc.

Healthy Mothers, Healthy Babies Coalition

March of Dimes

National Association for the Education of Young Children

National Black Women's Health Project

National Center for Education in Maternal and Child Health

National Maternal and Child Health Clearinghouse

Safe Kids Coalition

Health Education, Information, and Promotion Organizations

American Hospital Association, Health Promotion Center

American Dietetic Association

American Lung Association

American Red Cross

Association for the Advancement of Health Education

Consumer Health Information Resource Institute

Consumer Information Center

Environmental Defense Fund

Health Education Center

Health Education Foundation

Health Insurance Association of America

Health Media Education

HealthWorks Northwest

The Henry J. Kaiser Family Foundation

National Health Information Center

The National Health Network

National Information System for Health Related Services

National Public Health Information Coalition

Patient Education Resource Center

Society for Public Health Education

Women's Occupational Health Resource Center



Table 2 (continued). Other Organizations that Would be Interested in Educating the Public About Childhood Lead Poisoning

Civic Organizations

Federation of Women's Clubs
Kiwanis
Knights of Columbus
League of Women Voters
Shriners
Young Mens' Christian Association
Young Womens' Christian Association

Housing and Finance Organizations

Association of Local Housing Finance Agencies
Building Owners and Managers Association

Council of State Governments

Federal National Mortgage Association

Housing Assistance Council

Mortgage Bankers of America

National Apartment Association

National Association of Counties

National Association of Governments

National Association of Home Builders

National Association of Housing and Redevelopment Officials

National Association of Realtors

National Community Development Association

National Council of State Housing Agencies

National Council of State Legislatures

National Housing Conference

National Leased Housing Association

National Low Income Housing Coalitions

Advocacy Groups

Alliance to End Childhood Lead Poisoning
American Association on Mental Retardation
Association for Retarded Citizens of the United States
American Federation of Teachers
Child Welfare League of America
Children's Defense Fund
Citizen's Clearinghouse for Hazardous Waste
Coalition on Human Needs

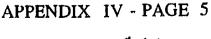




Table 2 (continued). Other Organizations that Would be Interested in Educating the Public About Childhood Lead Poisoning

Advocacy Groups (continued)

Foundation for Child Development
The Lead Coalition
Legal Services Corporation
National Association for Rights, Protection, and Advocacy
National Education Association
National Parent-Teacher Association

Artist Safety Organizations

Arts, Crafts and Theatre Safety (ACTS)
Center for Safety in the Arts

APPENDIX V

INFRASTRUCTURE DEVELOPMENT FOR ABATEMENT OF LEAD HAZARPS IN HOUSING

In the past two decades progress been limited in reducing childhood lead poisoning caused by lead-based paint and dust in homes. Only a small fraction of the housing units with lead-based paint have had the lead abated. To make matters worse, improper techniques were used in many past abatement projects. The high levels of lead in dust generated during abatement sulted in poisoning of workers and their families, and children left in their homes and abatement had exacerbations of lead poisoning. Inadequate abatement and cleanup procedures also resulted in children being repoisoned upon returning to their "deleaded" homes.

Great strides have been made in the past few years in improving abatement technology and protecting workers and their families. Although further improvements in abatement technology and practice are needed, we now have the tools to start a national abatement program. This section details the steps that must be taken to increase the national capacity to do safe and effective abatement work.

GUIDELINES DEVELOPMENT

The first set of comprehensive technical guidelines for lead-based paint testing and abatement, developed by a committee of government and nongovernment experts, were issued on an interim basis in April 1990, by the Department of Housing and Urban Development (HUD) for public and Indian housing authorities (the HUD Interim Guidelines). The HUD Interim Guidelines emphasize lead abatement of large blocks of units at the same time that other renovation work is done (comprehensive modernization). These guidelines were developed for housing that is to be extensively modified during modernization by the Federal government. These guidelines must be modified for use by States, localities, and individuals in situations where funds are scarce, time is critical, or the unit is not being gutted for other reasons.

WORKER TRAINING AND CERTIFICATION

Worker Safety

Lead-based paint abatement is a potentially hazardous occupation. Exposures among abatement and other workers, especially among pregnant women and among women and men who have or are planning to have children, should be reduced. Currently, lead abatement workers are not covered by the Occupational Safety and Health

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Administration (OSHA) general-industry standard regulating worker exposure to lead. Instead, they are covered under the safety and health standards for the construction industry, which regulate lead exposure less strictly. A standard is needed that takes into account new data showing adverse effects of lead on adults at levels well below the current OSHA general industry standard. Abatement workers should be protected by medical monitoring and medical removal provisions, as are potentially lead-exposed workers in general industry. Since many companies performing abatement are likely to have only a few employees, all companies, regardless of size, must be required to conform with Federal standards.

Curriculum Development

Federally developed or sanctioned model training programs are a method for assuring the quality and consistency of worker training. Basic course curricula must be developed to meet the training needs of different groups: HUD staff, public housing authorities, individual homeowners and landlords, contractors, workers, architects, designers, testers, and inspectors. Since such courses are a prerequisite for all other training activities, developing these course curricula should be given highest priority. Some curriculum development has already begun for implementation of the HUD Interim Guidelines.

Course Delivery Mechanisms

As the amount of lead paint abatement increases, market forces will meet the growing demand for training programs. In the short term, however, government involvement may be necessary. One option, establishing government-funded pilot training centers, was used successfully to deliver training to asbestos workers quickly. This approach offers a high degree of quality control and assures that training is available in all geographic areas. Pilot training centers, however, are expensive and could discourage centers without government funding from entering the market. Alternatively, the government could establish core curricula or curriculum requirements for each course. Federal or State governments or some other group could then evaluate private instruction programs and certify their adequacy. This approach would encourage the immediate involv of universities, labor organizations, and others and would probably provide the greatraining capacity in the long run. Although low in cost, this alternative does require government personnel or contract staff to review and approve each training program. In any event, mechanisms to control the quality of instruction and assure the competence of trainees are essential.

Certification

Institutionalizing lead paint abatement training will be difficult without mandatory requirements for certifying contractors and their workers, testers, and inspectors. At a minimum, individual training programs must be approved by a Federal or State agency or some other body, such as a trade organization. The Environmental Protection Agency



(EPA) used this approach--certifying training materials and approving course providers--in the initial phase of its asbestos program. Another alternative is for the certifying body to require that workers simply pass a standardized test. EPA is using this general approach to license radon-testing personnel. A third approach is performance-based accreditation, as in Massachusetts.

Institutionalizing Abatement Training

Lead-based paint abatement will probably not evolve exclusively as a separate industry and skill speciality. Lead-based paint abatement is an integral and inevitable part of a variety of existing building trades: painting, plastering, masonry, flooring, cabinetry, carpentry, electrical, plumbing, insulation, and door and window replacement. Therefore, lead-based paint abatement should be integrated into the various building trades, and all workers involved in home renovation and repair should be familiar with the special safeguards and techniques required.

A potential benefit of a national abatement program is increased employment. Most of the neighborhoods that will be targeted for lead abatement have high unemployment rates. As persons with little training develop the skills needed for leaded-paint abatement, they are likely to leave jobs that do not require training. Because this abatement work will require a large work force, training and employing local persons will have local economic and social benefits.

LABORATORY ACCREDITATION

Although laboratory testing protocols and quality assurance mechanisms currently exist for analysis of lead in air, water, and blood, no similar program, either mandatory or voluntary, exists for the analysis of lead in paint film or dust. Currently, EPA is distributing detailed instructions on standard test procedures for laboratories. However, within the next 18 to 24 months, some laboratory accreditation program is clearly needed to assure that consistent and reliable laboratory results are obtained. Options include a direct Federal laboratory certification program, a new independent voluntary accreditation program, or an expansion of existing accreditation programs for analyzing lead in other media to include tests of paint and dust.

EVALUATING EMERGING ABATEMENT TECHNOLOGY

During the past few years, private firms have developed a variety of new products to reduce the costs of lead-based paint abatement. Currently, more than a dozen new encapsulants and chemical strippers are being marketed across the country. Unfortunately, few independent standards have been developed or tests conducted to evaluate the effectiveness of these products or substantiate the claims made by manufacturers. Standards must be set and performance criteria established to assure the effectiveness of emerging products, either by the Federal government or by



nongovernment consensus. Such standards would allow private laboratories to test new lead abatement products at the manufacturer's or vendor's expense.

DISPOSAL OF ABATEMENT DEBRIS

At present, a significant impediment to broad scale abatement of lead-based paint in housing is uncertainty about whether the debris generated can go in regular municipal landfills as solid waste or must be disposed of as hazardous waste, at substantially greater expense. When lead is removed from buildings, it is, in effect, being concentrated; therefore, rules and regulations for its safe disposal are critical to prevent widespread dispersal throughout the environment. Although at present disposal is not an issue for the individual homeowner because of a household exemption, it is a problem for society. Certain wastes, such as stripping agents and cleanup materials with high dust concentrations, may be subject to hazardous waste classification and disposal requirements. Most abatement debris, especially bulky items, such as old window and door frames painted with lead-based paint, are likely to be considered not hazardous. Nevertheless, contractors are having difficulty finding laboratories to do toxicity testing, and insurance companies are wary of these requirements which place the responsibility and burden of proof on the unit owner and contractor. The situation is further confused by the conversion to a new toxicity test method planned for the summer or autumn of 1990. As soon as requirements for the new toxicity tests are finalized, clear and practical guidance must be given to contractors and owners of multifamily units as to how they should segregate waste debris so that as much of the debris as possible will not be classified as hazardous.

RELOCATION DURING ABATEMENT

Under most circumstances, residents and their pets should not occupy their housing during abatement. One of the most serious problems faced by local abatement programs is the lack of suitable temporary housing for families while their homes are being abated. Although relatives and friends have traditionally provided such housing, consideration should be given to special provisions in government-subsidized or other housing programs to deal with this special problem. Since most abatement projects take only a week or two, each unit provided for relocation purposes could be used for 25 to 50 families per year.

INSURANCE FOR CONTRACTORS

Another constraint to rapidly expanding lead-based paint abatement programs is the lack of insurance for contractors and building owners performing abatement work. As in the case of asbestos, improper abatement techniques used in the early years raised concerns among insurance companies about providing liability coverage. With the availability of guidelines on safe practices, the marketplace can be expected to respond with coverage at reasonable prices. Comprehensive coverage is already being provided by the



self-insurance risk retention pool established by many large public housing authorities. It is hoped that private insurers will soon recognize this market and provide coverage at competitive rates. Federal, State, and local agencies should take steps to encourage or require such coverage.

